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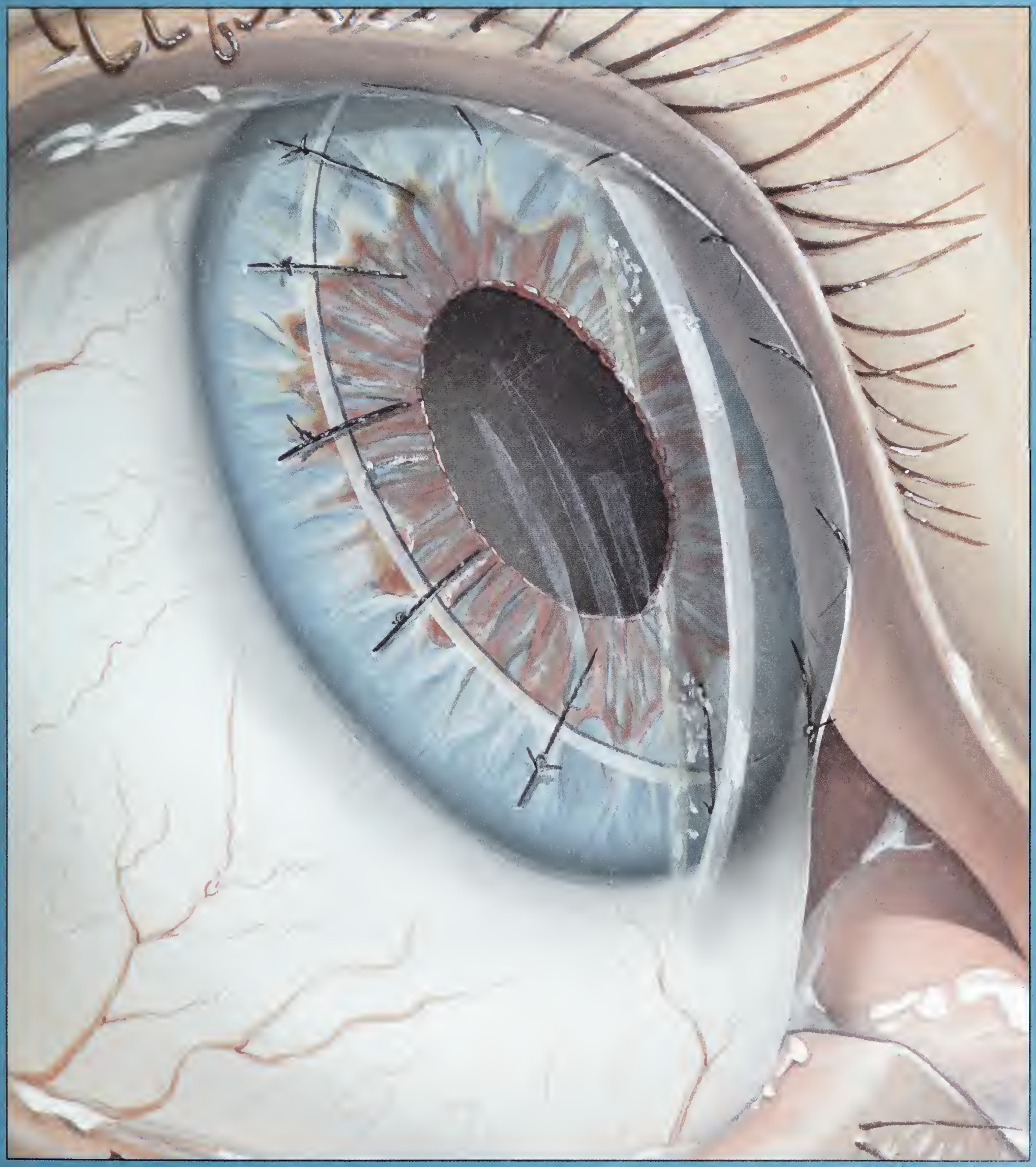

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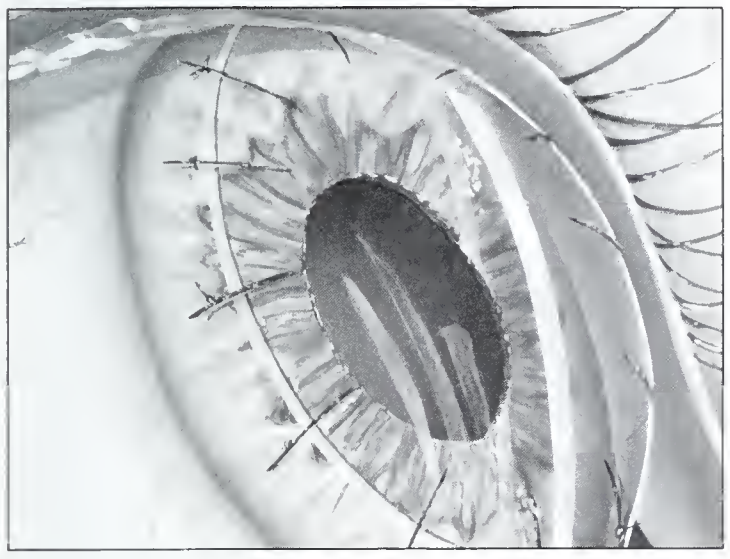

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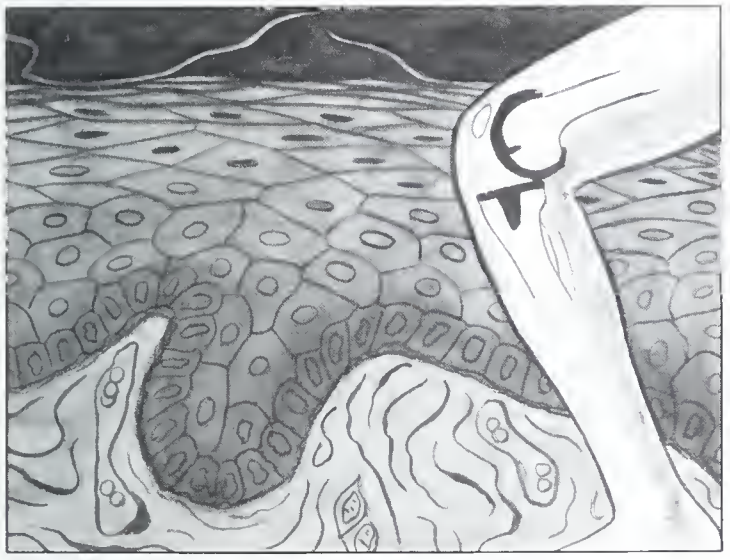

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Editorial

Dear Readers,

We are happy to say that the Journal has entered its seventieth successful year of publication. As the new editors, we feel a certain sense of awe at having been chosen to follow in the footsteps of such a longstanding tradition. It is, indeed, a privilege and an honour. The last seventy years of publication have seen changes in the field of medicine that were far beyond anything that our predecessors would have thought possible. If they had had a telescope that allowed them to view the future, they may have been encouraged to know that much of their efforts in research and the implementation of that knowledge in the care of their patients laid the foundation for many of the astounding advances that we take for granted.

Our only hope is that the next seventy years will be witness to as much, if not more, progress in the medical field. Here at the University of Toronto Medical School, research has a prominant place. We are proud to be a part of an institution that recognizes the importance of being at the forefront of the quest for new knowledge. Medical research is something that is close to the hearts of both of us. Without it, medicine becomes stagnant and introverted. To have no interest in research, is to be satisfied by the status quo. As long as there is sickness, there will be a need for medical research. There is a part to play for all of us as physicians. If we cannot participate directly, we at least owe it to ourselves and our patients to keep abreast of the latest research discoveries. That is, in fact where "the rubber meets the road", the interaction between patient and physician. As such, we feel strongly about keeping you informed of the research opportunities that our students have engaged in. It is with great pleasure that we bring you this issue of the UTMJ.

Sincerely,

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A Review of Corneal Transplantation: Historical Perspectives and Future Directions

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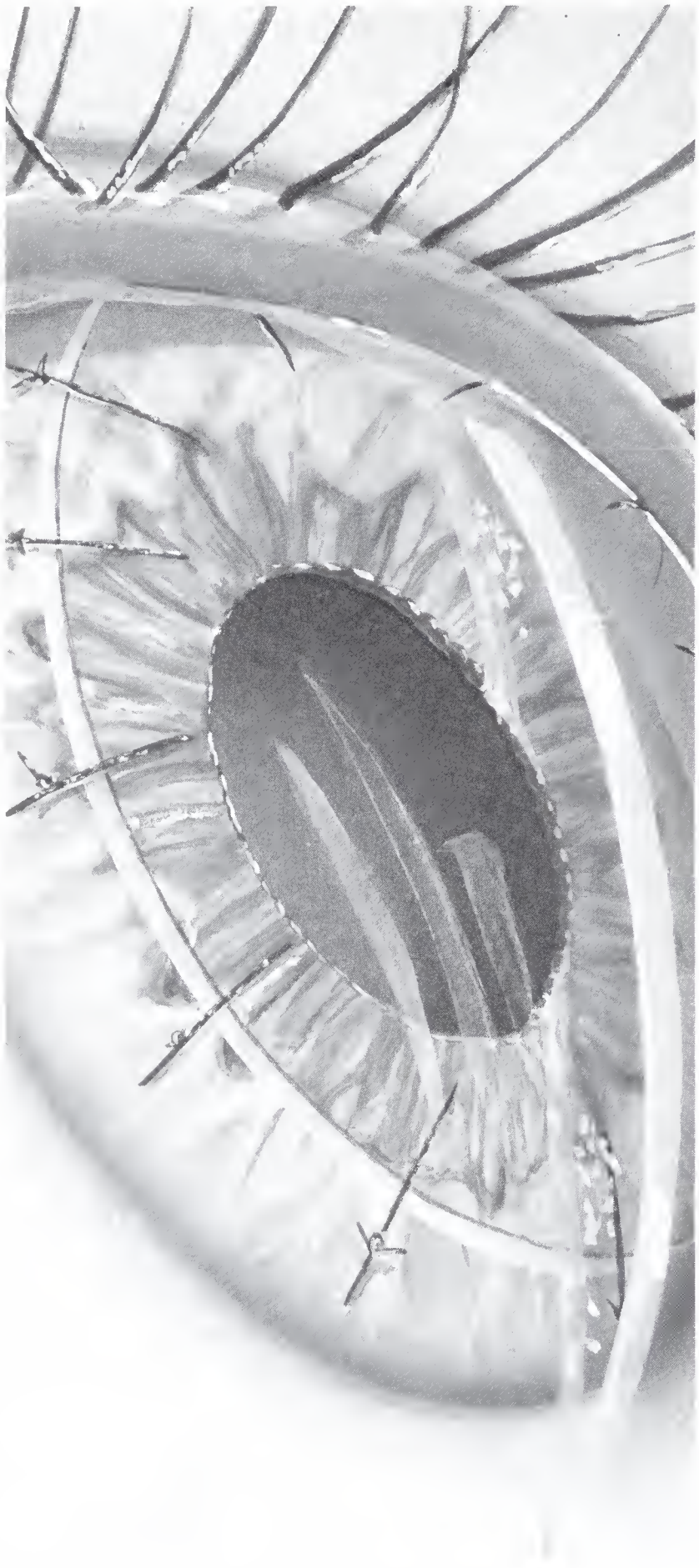


LORETTA DE GROOT

Abstract

Keratoplasty is the most successful organ transplant procedure performed, and each year preserves sight for thousands of Canadians. The Eye Bank of Canada supplies most of the corneas used in transplantation, with the Ontario Division using an elective scheduling procedure. However, it is important to educate the public to the increasing need for donor corneas. Most corneal grafts provide excellent visual results, with the majority of failures resulting from allograft rejection. Research in the area of HLA matching and immunosuppressive drug therapy shows promise for improving graft outcome in high-risk patients.

Introduction

In the late 18th century, the French researcher Guillaume Pellier de Quengsy first suggested replacing an opacified cornea with a thin glass disc set in a silver ring and stitched to the sclera[1]. However, Pellier de Quengsy never performed the operation, and it was left to S.L.L. Bigger who, in 1835, performed a successful homograft on his pet gazelle while both were held captive by Sahara Bedouins[2]. In the late 19th century, the work of Arthur von Hippel initiated a new era in keratoplasty with his appreciation of corneal biology, and his invention of the circular trephine, the mainstay of current keratoplastic instrumentation. The first full thickness corneal graft to remain clear in humans was performed by Edward Zirm in 1906[1]. In this famous case, the donor cornea was obtained from an 11-year-old boy whose eye had been injured by a foreign body, and the recipient was a man who had been blinded by lime burns. There was partial success with restoration of vision in the left eye to 6/36.

The modern era of keratoplasty began in the 1950s with the advent of microsurgery, fine instruments and sutures, the establishment of eye banks and the development of anti-inflammatory and immunosuppressive drugs to control graft rejection. Keratoplasty is now the most widely practiced form of clinical transplantation, with 2,481 transplants performed in Canada in 1991[3].

Anatomy of the Cornea

The cornea has two major functions: protection of intraocular contents and refraction of light (the cornea

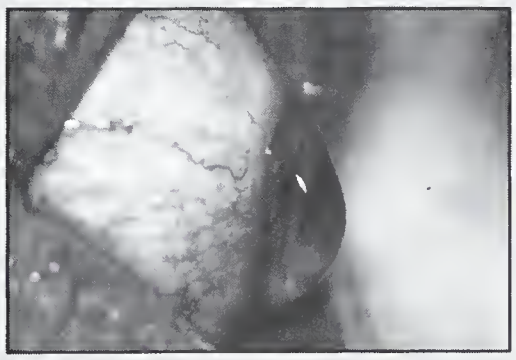
provides 66% of the eye's refractive power). The cornea consists of five layers: the stratified squamous epithelium (which blocks penetration of water into the stroma), Bowman's membrane, the stroma (which provides 90% of the cornea's thickness), Descemet's membrane, and the endothelium. The endothelium maintains corneal transparency by keeping the stroma dehydrated[4]. The cornea is an avascular tissue, receiving its nutrients from tear film, aqueous humor and limbal vessels.

Types of Keratoplasty

There are two types of corneal transplant procedures. Lamellar keratoplasty is defined as removal and replacement of less than the total thickness of the cornea[5]. The host's Descemet's membrane and endothelium remain intact and serve as the base onto which the donor tissue is grafted. Lamellar keratoplasty is indicated for those corneal conditions in which there are anterior stromal or surface irregularities but the endothelium is intact, as is the case with superficial scars or surface irregularities caused by stromal or surface disease[4]. However, lamellar keratoplasty does not yield visual results comparable to penetrating keratoplasty, and hence is primarily used to strengthen and thicken the corneal substance in anticipation of subsequent full-thickness keratoplasty[6].

Penetrating keratoplasty, in which a full-thickness cornea is transplanted, is necessary when the primary defect is in the corneal endothelium or when the stroma is deeply opacified or thinned (Fig. 1). The procedure involves removing the patient's cornea with a trephine under local anesthesia and replacing it with a donor cornea of similar or slightly larger size, usually 7.0-8.5 mm in diameter. More than 90% of corneal transplants are of this type[6]. The major indications for penetrating keratoplasty are: (1) keratoconus (a noninflammatory, usually bilateral protrusion of the cornea) (Fig. 2), (2) pseudophakic bullous keratopathy (corneal degeneration following cataract extraction and intraocular lens implantation), (3) aphakic bullous keratopathy (corneal degeneration following cataract extraction without intraocular lens implantation), (4) Fuch's dystrophy (an endothelial dystrophy that affects the central cornea resulting in edema) (Fig. 3), (5)

Figure 1



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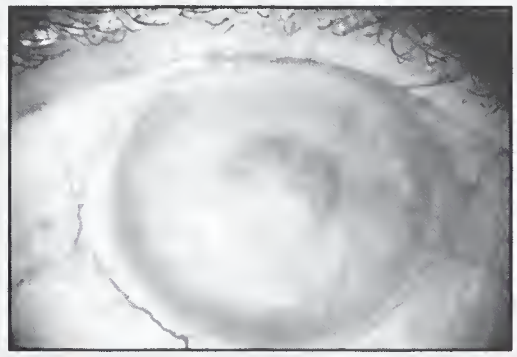

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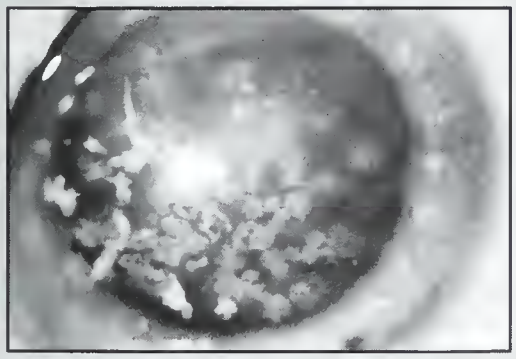

Figure 4: Granular dystrophy, a hereditary stromal dystrophy, with multiple white deposits in the central cornea.

Figure 1: Status-post penetrating keratoplasty with the sutures out and a clear graft.

Figure 2: Keratoconus with a conical-shaped cornea and irregular astigmatism.

Figure 3: Fuch's corneal dystrophy with diffuse edema and superficial scarring.

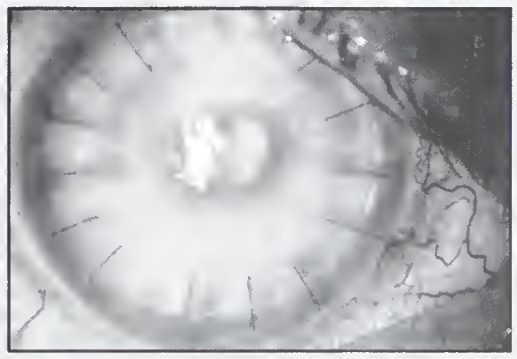

Figure 5: Graft rejection with inflammatory cells on the endothelium and diffuse swelling. Treatment with topical steroids resulted in complete clearing of the graft.

regrafts (repeated penetrating keratoplasty following previous graft failure) and (6) scars (secondary to infections, trauma, or hereditary stromal dystrophies) (Fig. 4) The relative frequencies of these indications continues to change. For example, pseudophakic bullous keratopathy will probably decrease in incidence as more surgeons utilize posterior rather than anterior lens implantation following cataract extraction[6].

Refractive keratoplasty has the goal of using corneal grafts to change the curvature of the patient's cornea and hence correct optical errors. The most widely practiced of these procedures is epikeratophakia, in which the donor cornea is carved on a cryolathe and placed on the patient's Bowman's layer[12]. Epikeratophakia is used for aphakia, myopia, and keratoconus. As these procedures become more refined and thus more widely used, it is anticipated that the need for donor corneas will be greatly increased.

The Eye Bank of Canada

The Eye Bank of Canada is a joint project of the University of Toronto and the Canadian National Institute for the Blind (CNIB) which obtains pledges for eyes and arranges transportation of donor eyes to the Eye Bank. One may become an eye donor by signing an eye bank donor card, or any other donor consent form such as the back of the Ontario driver's licence. Another important means of obtaining corneal tissue is by approaching the next-of-kin at the time of death. Approximately one half of the corneas obtained are used for transplantation, the rest being used for teaching and research. Although the number of corneas being donated has increased yearly since the Eye Bank was established in 1955, a waiting list of potential recipients exists for most provinces, with the waiting time ranging from three months to greater than one year. However, the Ontario Division is the only eye bank in Canada which uses an elective system. The surgeon books the surgery and the tissue in advance, and it is uncommon for surgery to be cancelled due to lack of tissue[13]. This is in contrast to the traditional systems used for transplants where one cannot book surgery until it is known that there is tissue available. The use of new grafting techniques in refractive surgery will undoubtedly increase the demand for donor tissue. This increased demand is contrasted with a public attitude which is currently an impediment to procurement of body parts for transplantation. A recent report in the Canadian Journal of Ophthalmology found that while 63% of those surveyed were willing to donate body parts, only 37% had signed either the organ donor portion of their driver's licence or an organ donor card[14]. Clearly, more public education is needed to advocate the benefits of corneal transplantation, the need for donor corneas, and the process of eye donation.

Obtaining, Preserving & Screening of Donor Tissue

Cadaveric corneas are obtained from donors up to twelve hours after death. The cornea may then be stored in a moist chamber, in which case it must be transplanted within 24-48 hours. However, most corneas are placed in a preservative medium such as the McCarey-Kaufman (MK) medium which allows storage for up to four days[15]. More recent developments include the use of a chondroitin-sulfate medium which allows storage for up to one week. This provides time for transport of corneas over long distances and decreases waste of stored corneas.

In addition, advances in corneal preservation have allowed eye banks time to evaluate the quality of the donor cornea more thoroughly. Not all corneas harvested are suitable for transplantation either due to underlying donor disease or poor donor quality. Many CNS, infectious, malignant, and intrinsic eye diseases are absolute contraindications for surgical use of the donor cornea. Corneas from donors with known or suspected high risk for HIV infection are excluded from surgical use regardless of serotyping results. In addition, HIV is screened for routinely in all donor corneas using ELISA.

The quality of the donor tissue is critical to the success of the graft. The endothelial layer is acknowledged to be of fundamental importance, and this may be evaluated by specular microscopy, which allows assessment of endothelial cell density and morphology[16]. The association between donor age and graft success is less clear-cut[17], although tissue from a young donor is preferable, particularly for a young recipient.

Complications of Keratoplasty

The goal of keratoplasty is to provide a clear graft and hence improve vision; this is achieved in more than 90% of select cases[18]. Despite excellent results, graft failure remains a significant problem with penetrating keratoplasty. There are many causes of graft failure. Surgical complications, persistent epithelial defects, infection, glaucoma, trauma, and recurrence of primary corneal dystrophies are common etiologies[19].

The leading cause of late graft failure is immune-mediated allograft rejection (Fig. 5)[20]. In optimal cases, the inci-

dence of graft rejection is approximately 30%[6], but in high-risk cases, as with previous graft failure or extensive deep corneal vascularization, rejection may occur more than 60% of the time. Many graft rejections can be reversed if treated early with intensive corticosteroid therapy. However, approximately half cannot be reversed and lead to graft failure. Strategies for suppressing the corneal allograft response include reduction of antigenic difference via HLA matching, and aggressive immunosuppression with newer drugs such as cyclosporin A.

Class I HLA antigens, which are known to play an important role in graft rejection, have been demonstrated on corneal epithelial, stromal, and endothelial cells[21]. Several studies[22-23] have demonstrated a lower incidence of graft rejection when at least two donor and recipient HLAs are matched. In a series of penetrating keratoplasties with high rejection risks, Sanfilippo *et al.* found that 49% of patients with a poor HLA match experienced graft rejection, compared to only 21% of those with a good HLA match. However, class I matching does present some logistical difficulties. Tissue typing is best done on fresh lymphoid tissue (spleen or lymph nodes), but this is seldom available from the usual pool of corneal donors[24]. Furthermore, the HLA system is very polymorphic and a large pool of donors and recipients is necessary if good matches are to be found.

Immunosuppressive drugs also play an important role in preventing graft rejection. Topical corticosteroids are universally employed in the post-operative management of corneal grafts. However, their frequent side effects, which include raised intra-ocular pressure and cataract formation, preclude the long-term, high-dose use required to prevent rejection in high-risk cases. Cyclosporin A is an extremely potent immunosuppressive that is used in major organ transplantation. Studies using animal models have shown systemic administration of cyclosporin to prolong graft survival[25]. Topical preparations have not been shown to be as effective as anticipated[26]. The high cost of the drug and its major side effects (nephrotoxicity, hypertension) will probably limit its use in the prevention of corneal graft rejection.

Summary

Keratoplasty is a highly successful procedure in which diseased corneal tissue is removed and replaced by donor corneal material. The concept of corneal transplantation is about 200 years old, but the modern era of keratoplasty began in the 1950s with advances in both surgical technique and pharmacology of anti-inflammatory drugs. While keratoplasty was quite uncommon 40 years ago, nearly 2,500 transplants are now performed in Canada yearly. Ninety percent of keratoplasties performed are penetrating keratoplasties, which are indicated in conditions with endothelial involvment or deep stromal opacification. The Eye Bank of Canada supplies most of the corneas used in transplantation. The Ontario Division utilizes an elective scheduling procedure. The public must be educated to ensure the increasing demand for donor corneas will be met in the years to come. Most keratoplasties yield clear grafts and improved vision. Immune-mediated allograft rejection is the leading cause of late graft failure. Reducing antigenic difference via HLA matching and new immunosuppressive drugs are areas of intense research that show promise of improving the prognosis of high-risk cases.

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Corrections

In Vol. 69, No. 3, the following corrections should be noted. In *The medical student's guide to facial fractures,* the pen and ink line work for figures 1, 5, and 6 were graciously drawn by Loretta De Groot, 9T4. In the Article, *Assessing competency to consent to treatment: A review and guide,* the painting on page 22 was produced by Mara Korkola, not Marc Korkola. Our apologies to the Otolaryngology Department at Mt. Sinai Hospital for misrepresenting their name and to Dr. Ruth Kurdyak of the Medical Alumni Association for not correctly identifying her year of graduation as 6T0.

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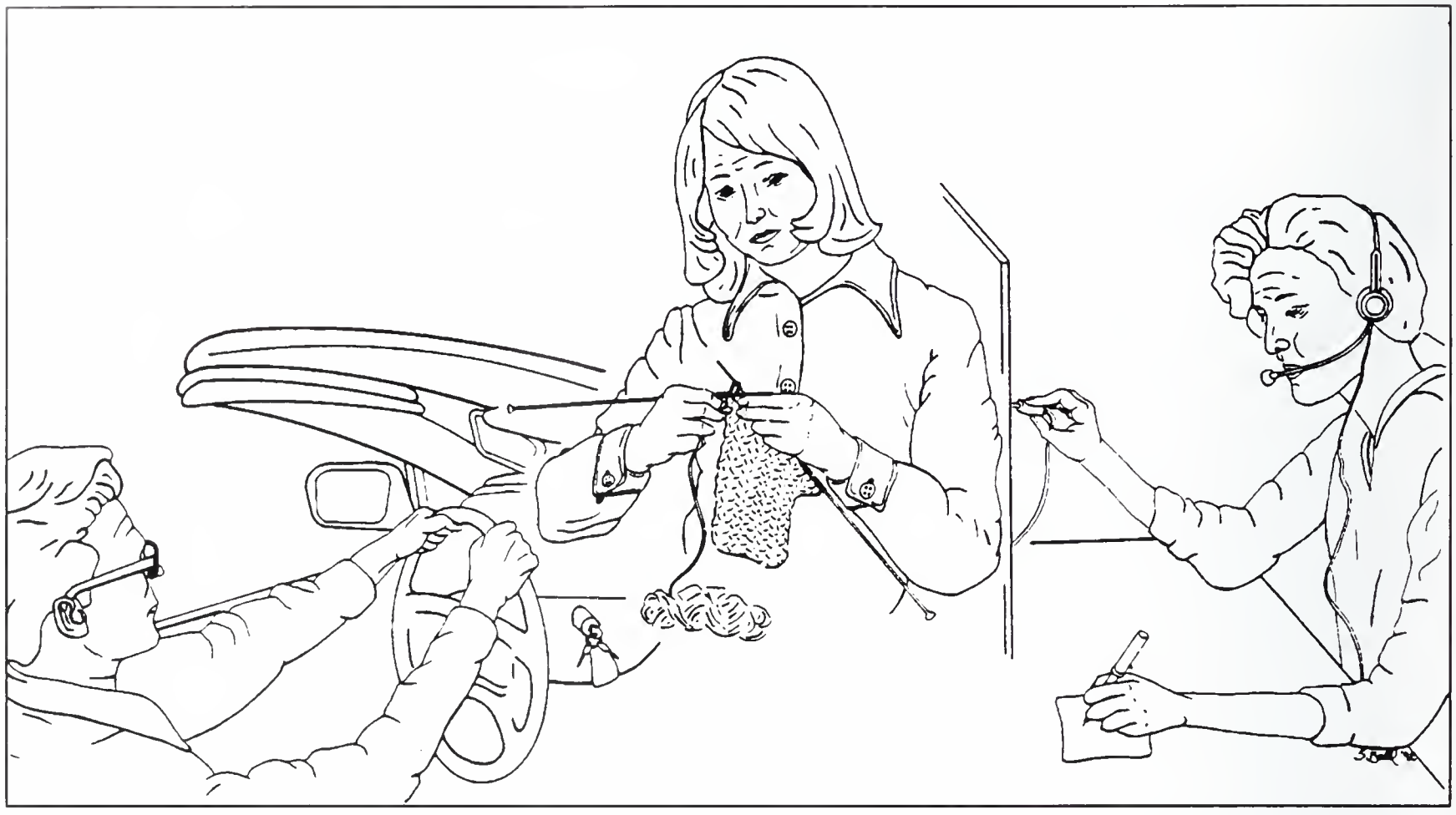

SUSAN BALL

Compression Neuropathies of the Upper Extremity

Suzanne R. Strasberg B.Sc., M.D. and Richard Korentager M.D., F.R.C.S.(C).*

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Abstract

Compression neuropathies of the upper extremity are frequently encountered problems which continue to be disabling. The associated morbidity frequently results in enormous personal and financial loss. Proper and prompt diagnosis and treatment is necessary to reduce this associated morbidity. The purpose of this paper is to discuss the pathogenesis, diagnosis and treatment of the three most common compression neuropathies of the upper extremity: carpal tunnel, ulnar tunnel, and cubital tunnel syndromes so that recognition may be early and effective care may be given promptly.

Compression neuropathies of the upper extremity are extremely common problems which affect thousands of individuals yearly. Their associated morbidity is responsible for enormous economic and personal losses. The purpose of this paper is to discuss the similarities and differences in pathogenesis, diagnosis and treatment of the three most common compression neuropathies affecting the upper limb: carpal tunnel, ulnar tunnel and cubital tunnel syndromes.

Pathogenesis

The three nerve compression syndromes are similar in that they occur as the nerve passes through natural anatomic tunnels. Compression occurs when the cross-sectional area of the tunnel is reduced. This can result from extrinsic compression of the tunnel, narrowing of the tunnel itself or an increase in the tunnel contents, either by enlargement of a natural content or by a space-occupying lesion. The neurologic abnormalities are believed to be related to several factors including arterial insufficiency, intraneural edema and intraneural fibrosis. Mechanical deformation of the nerve itself may also be a contributing factor[2,3].

There are numerous etiologic factors responsible for entrapment neuropathies which may be best remembered by considering disease processes (SEE TABLE 1).

Anatomy

Carpal tunnel syndrome (CTS) was first described in 1854 by Paget (For review, refer to reference #1). It con-

Table 1: Causes of Entrapment Neuropathies

CAUSES	EXAMPLE
Congenital Narrowing of the Tunnel	
Proliferative Disease	Any chronic arthritis e.g., rheumatoid arthritis, scleroderma, SLE
Infiltrative Disease	Amyloid; ganglia; anomalous muscles; tumors
Metabolic	Gout; pregnancy; obesity
Endocrine Disease	Diabetes mellitus; hypothyroidism; acromegaly
Trauma	Wrist, forearm and elbow fractures; repetitive use of hands, fingers, and elbow; use of vibrating tools
Acute Crowding of the Tunnel	Infection; fractures of wrist and forearm
Idiopathic	

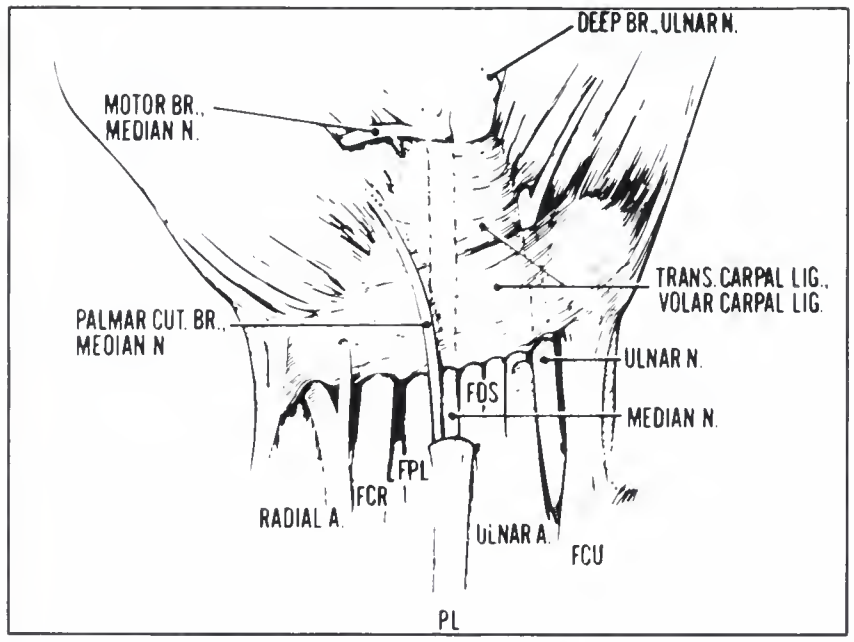

Figure 1: Anatomy of the carpal and ulnar tunnels FCR – Flexor carpi radialis, FDS – Flexor digitorum superficialis, PL – Palmeris longus, FCU – Flexor carpi ulnaris, FPL – Flexor pollicis longus.
(From Greene DP (Ed): Operative Hand Surgery. Vol 2. Churchill Livingstone, 1982).

sists of a constellation of symptoms including numbness, paresthesiae, pain and weakness of the median innervated structures of the hand. The median nerve is predisposed to mechanical compression because of its passage through an unyielding anatomic space. This space, known as the carpal tunnel, is bounded medially, laterally and posteriorly by carpal bones and superiorly by the transverse carpal ligament (flexor retinaculum)[2]. Through this space run the nine extrinsic flexor tendons, their synovium, the median nerve and its blood supply (Fig. 1).

Ulnar nerve compression at the wrist as it courses through the ulnar tunnel (Guyon's canal) is known as ulnar tunnel syndrome (UTS). The ulnar tunnel begins at the proximal end of the pisiform bone and ends distal to the hook of the hamate. The floor of the tunnel is formed by the flexor retinaculum. The roof is made of the volar carpal ligaments proximally. Distally it is formed by fibres of the palmar fascia and palmaris brevis[4] (Fig. 1).

Two types of UTS have been described. Type 1 is associated with compression of the sensory and motor fibres at the level of the pisiform bone just proximal to where the deep branch of the ulnar nerve exits the main trunk. Type 2 UTS involves only compression of the deep motor branch of the ulnar nerve, just distal to the hook of the hamate[4].

Cubital tunnel syndrome (CuTS) occurs with entrapment of the ulnar nerve as it passes through the cubital tunnel at the elbow. Cubital tunnel anatomy can be divided into 3 parts: 1) the entrance of the tunnel which begins at the ulnar groove just posterior to the medial

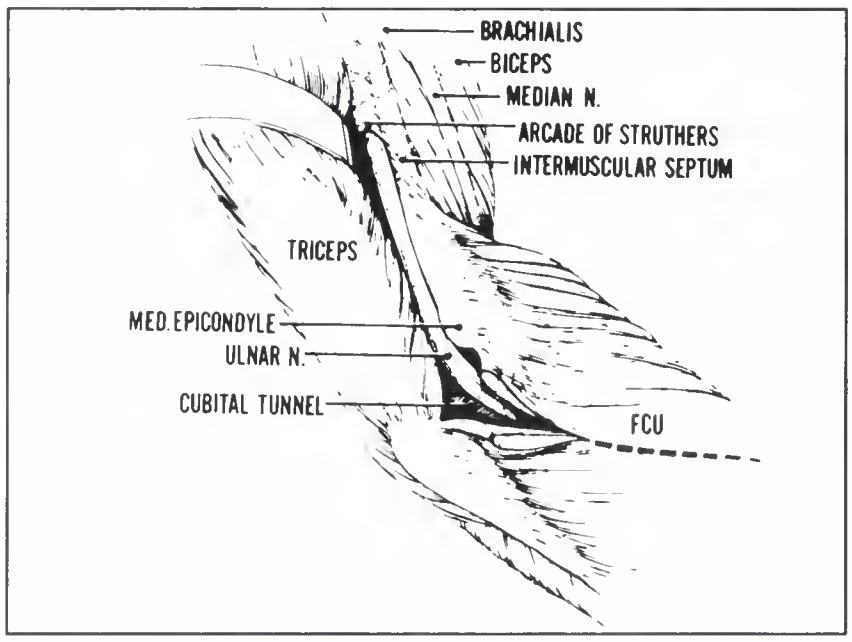

Figure 2: Anatomy of the cubital tunnel FCU – Flexor carpi ulnaris.
(From Greene DP (Ed): Operative Hand Surgery. Vol 2. Churchill Livingstone, 1982).

condyle of the humerus, 2) the fascial aponeurosis which joins the two heads of the humerus, and 3) the muscle bellies of the flexor carpi ulnaris (FCU)[3]. It is bordered medially by the origin of the flexor carpi ulnaris heads and laterally by the elbow joint itself[3] (Fig. 2). The ulnar nerve is a continuation of the medial cord of the brachial plexus. It runs medial to the axillary nerve and brachial artery until the middle third of the humerus where it pierces the intermuscular septum of the medial head of

the triceps[3]. Then it runs along the medial head of the triceps until the entrance to the cubital tunnel. It courses through the tunnel below the fascia which joins the two heads of the flexor carpi ulnaris and anterior to the flexor digitorum profundus (Fig. 2).

Diagnosis

History

An accurate history is the most effective diagnostic tool. One should obtain detailed information regarding the location, distribution and radiation of any pain, numbness or parasthesiae. All three conditions involve complaints of burning or tingling pain, numbness, hypoesthesia and weakness in the fingers and hands. Tenderness at the site of compression and along the course of the nerve may also be noted by the patient.

Patients with CTS complain of symptoms that typically involve their radial 3 1/2 digits. Weakness is usually manifested by complaints of clumsiness during the day or with activities requiring prolonged wrist flexion such as driving. Occasionally, patients complain of more proximal radiation of their pain[7] or shoulder pain only[6] as the presenting symptom. There is often a history of nighttime hypoesthesia waking the patient from his or her sleep[7].

Sensory symptoms of both CuTS and UTS involve the ulnar 1 1/2 digits[8,9]. Complaints of weakness may be reported as inability to perform minute finger movements due to weakness of the ulnar-innervated intrinsic muscles of the hand[4,8]. Patients with CuTS may present initially with only pain on the medial aspect of their forearm[3]. Patients with UTS type 1 will show a mixed picture of both sensory and motor symptoms, while symptoms of UTS type 2 are purely motor with no associated sensory deficits[4,9].

The patient should be questioned regarding his present occupation and hobbies, as well as those prior to the onset of symptoms. Fine repetitive movements of the wrist and fingers, especially those requiring prolonged wrist flexion as seen in typing or knitting, are associated with a higher incidence of CTS and UTS. Use of vibrating tools has also been implicated in carpal tunnel syndrome[7]. A higher incidence of cubital tunnel syndrome has been reported in patients with occupations or hobbies requiring repeated flexion of the elbow (e.g., telephone operators[8]).

Diabetes[1], gout[1], rheumatoid arthritis[9], thyroid disease, ganglia[4], tumors[1,8], pregnancy[2], past injuries and surgery to the upper extremity are predisposing or contributory factors and should be asked about in the systems review.

Review of current and past medications should include use of diuretics, NSAIDs, and local and systemic corticosteroids. Toxic neuropathies have been reported with some medications such as chemotherapeutic agents[7].

Physical

There are several signs which may be observed in entrapment neuropathies:

1. Hypoesthesia, dysesthesiae or paresthesiae may be found in the radial 3 1/2 digits in CTS and in the ulnar 1 1/2 digits in UTS and CuTS. Sensation in the dorsal sensory branch of the ulnar nerve distribution should be tested to help determine the level of entrapment. Ulnar nerve compression proximal to the wrist results in hypoesthesia in the dorsal aspect of the hand, while with ulnar nerve compression at the wrist, the dorsal sensory branch is spared.

2. Muscle weakness and atrophy. In CTS, atrophy occurs in the thenar muscles and may be obvious when present. Weakness may be elicited by testing the function of the abductor pollicis brevis muscle[2]. Atrophy of the hypothenar muscles and intrinsic muscles of the hand may be seen in UTS and CuTS[3,9]. This may be observed by testing the strength of the first dorsal interosseous muscle and adductor muscles of the hand. As well, weakness of the flexor profundus tendons to the ring and small fingers may be noted in cubital tunnel syndrome.

3. Tinel's sign is elicited by repeated light tapping over the nerve in the area of entrapment (i.e. over the carpal tunnel or cubital tunnel). The test is considered positive when a tingling sensation is elicited in the hand distal to the tapping in the distribution of the affected nerve[7]. A positive sign indicates nerve fibre irritation.

4. Phalen's sign is used to elicit symptoms of carpal tunnel and ulnar tunnel syndromes. It is performed by having the patient actively flex his wrist for 60 seconds. The test is positive if numbness is elicited in the median nerve distribution for carpal tunnel syndrome[7] and the ulnar nerve distribution for ulnar tunnel syndrome[4].

5. The elbow flexion test is used in cubital tunnel syndrome. The test is considered positive if increased numbness is noted in the ring and small fingers with full flexion of the elbow[3]. There may also be abnormal mobility of the ulnar nerve over the medial epicondyle of the humerus[10].

While the above signs should be sought, a full physical exam on every patient including a complete neurologic examination with evaluation of the upper extremity and cervical spine should be performed in order to rule out more proximal entrapment of the nerve or alternative diagnoses.

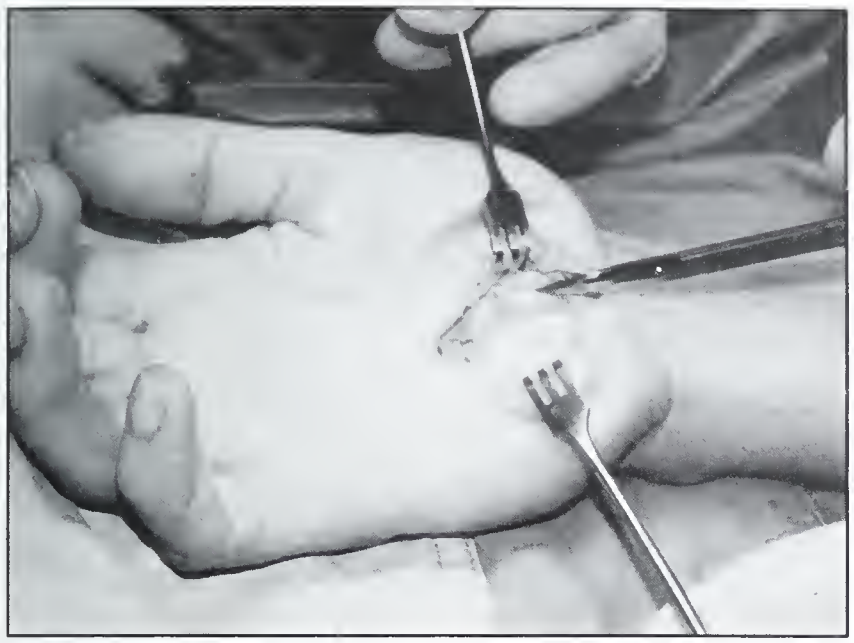

Figure 3: Median nerve in the carpal tunnel.

Ancillary Studies

While diagnosis of an entrapment neuropathy is almost always clinical, ancillary studies are important adjuvants to the clinical exam. While some advocate use of EMG and nerve conduction studies only when clinical exam is inconclusive[2,4], these tests are practically important for several reasons. Firstly, they provide a baseline by which one can assess post-surgical success or failure in the case of unresolved symptoms. Secondly, they may help to determine if a "double crush" or two co-existing areas of nerve entrapment are present. Thirdly, as many of these cases are work-related these tests are important when assessing a patient's disability for the Worker's Compensation Board or in the case of medico-legal difficulties. Finally, the results of these tests may help assess the timing of treatment. Patients with mild to moderate sensory deficits may be treated with conservative treatment. However, patients with motor nerve involvement are best treated with prompt surgical decompression.

Treatment

Management of all entrapment neuropathies may be divided into non-operative and operative.

Non-operative Management

Conservative treatment is indicated in patients with recent onset of symptoms, intermittent symptoms or those with no evidence of atrophy or denervation[2]. The cornerstone of conservative treatment in carpal tunnel and ulnar tunnel syndromes involves the static splinting of the wrist in the neutral position. This minimizes mechanical movement of the wrist, lowers the pressure within the tunnel and decreases shearing forces across the wrist[9]. Likewise, cubital tunnel syndrome requires immobilization of the elbow.

All three syndromes may be treated with a trial of NSAIDs or injection of corticosteroid into the affected tunnel[9]. Aggravating factors such as repetitive flexion of the wrist and fingers, use of vibrating tools or direct pressure over the elbow and wrist should be avoided[7,9].

Operative Management

The operative treatment of a compressed nerve involves surgical decompression of the nerve at the site of anatomical entrapment. This may be done under local, regional or general anesthesia.

Carpal tunnel syndrome

Surgical treatment involves release of the flexor retinaculum and decompression of the carpal tunnel. The usual skin incision is a palmar incision which follows the axis of the third metacarpal.

The palmar fascia is incised and the palmaris brevis muscle is often encountered. The flexor retinaculum is incised and the median nerve identified and protected (Fig. 3). The remainder of the flexor retinaculum and the antebrachial fascia are released. It is helpful to identify the recurrent branch of the median nerve to ensure that it is not separately compressed.

Postoperatively, the patient's wrist is immobilized with a dorsal slab for several days. Extension and flexion exercises of the thumb and fingers are encouraged.

Ulnar tunnel syndrome

Exposure of Guyon's canal for surgical decompression is obtained with a zig-zag incision along the radial border of the FCU across the wrist towards the fourth digit with care to avoid the ulnar palmar cutaneous branch[3]. This incision is very similar to that for carpal tunnel release and both procedures may be performed with the same incision if necessary. Once the ulnar aspect of Guyon's canal is released, the ulnar artery is sought and identified. The usual site of compression of the ulnar nerve is at the level of the pisiform bone[4]. If muscle atrophy or weakness is present, the deep branch of the ulnar nerve must be exposed as it runs between the piso-hamate ligament and the fibrous aponeurosis of the hypothenar muscle[4]. This is the most common location for deep branch entrapment[9].

Postoperative management consists of immobilization of the wrist for several days and avoidance of repetitive or heavy work for at least six weeks.

Cubital tunnel syndrome

Various techniques for surgical release of ulnar nerve entrapment in the cubital tunnel have been reported.

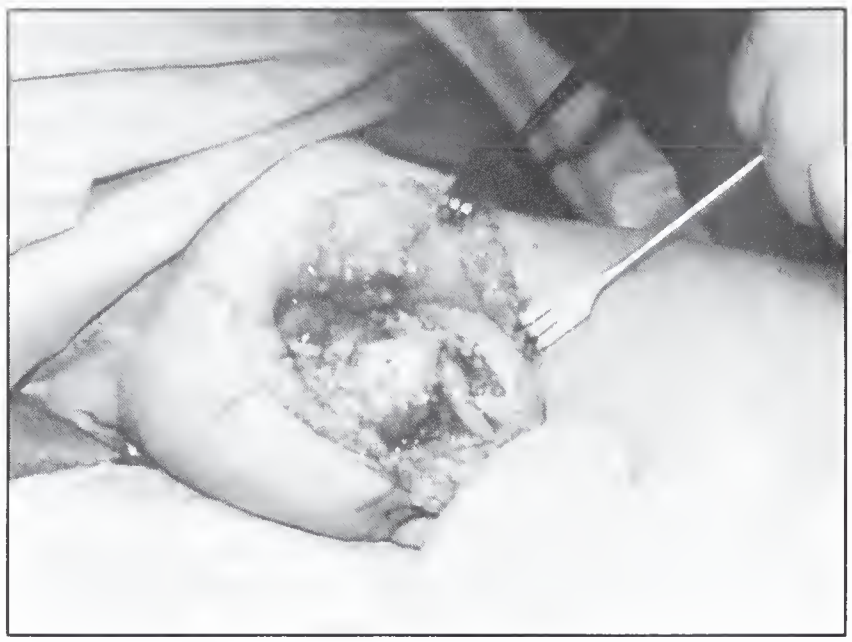

Figure 4: Submuscular transposition of the ulnar nerve at the elbow.

Surgical decompression begins with a curvilinear incision along the posterior-medial aspect of the elbow. The nerve is visualized proximal and distal to the cubital tunnel[9]. The aponeurotic bands of the FCU muscle overlying the nerve are cut and the nerve is traced distally to the proximal third of the forearm[3].

Anterior transposition of the ulnar nerve is often used in conjunction with simple surgical decompression (Fig. 4). This may be necessary in patients who continue to have compression of their ulnar nerve on elbow flexion or recurrent subluxation of the nerve over the medial epicondyle. Anterior transposition may be subcutaneous, subfascial or submuscular[8]. Following anterior transposition, secondary impingement is possible. It is important to avoid acute angulation of the ulnar nerve and to ensure that the medial intermuscular septum does not impinge on it[9]. Removal of this band, as well as release of the aponeurosis of the FCU may be necessary to avoid impingement[3].

Epicondilectomy was first described in 1950 by Morgan and King who proposed removal of the medial epicondyle instead of anterior transposition of the ulnar nerve (For review, refer to reference #3). Although this is a simple operation, it is not widely accepted any longer as it requires wide exposure of the nerve and results in a long disfiguring scar[8]. It also results in loss of the protecting prominence of the medial epicondyle which increases the susceptibility of the ulnar nerve to repeated direct trauma[3].

Postoperatively, the elbow is maintained in 90 degrees of flexion with a posterior splint and the wrist in a neutral position for three weeks[3]. Care must be taken to avoid bumping or reinjuring the nerve.

Complications

Results from surgery are excellent. However, there exists a small proportion of patients who fail to improve or who develop new symptoms following surgery[5]. The most common reasons include iatrogenic injury to, or inadequate release of, the entrapped nerve[5,11]; additional sites of compression along the nerve; incorrect diagnosis; and damage to an adjacent sensory nerve.[5,11,12]

Conclusion

Entrapment neuropathies are a common problem associated with significant morbidity. Knowledge of the common presenting patterns and treatment modalities are necessary for effective care of these afflicted patients.

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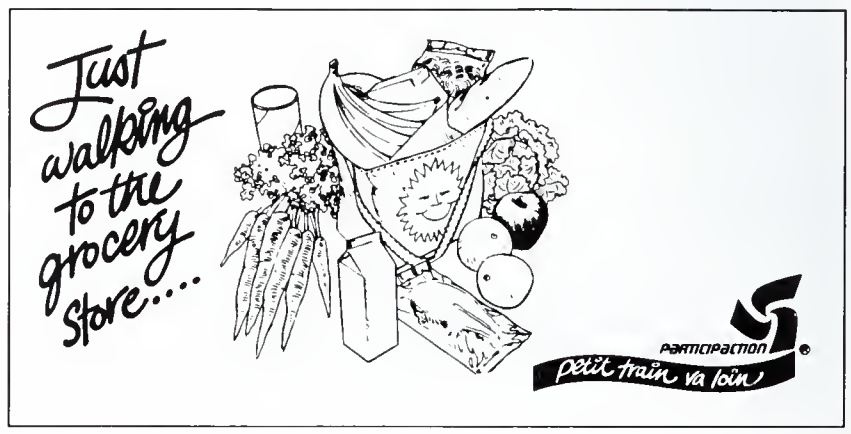

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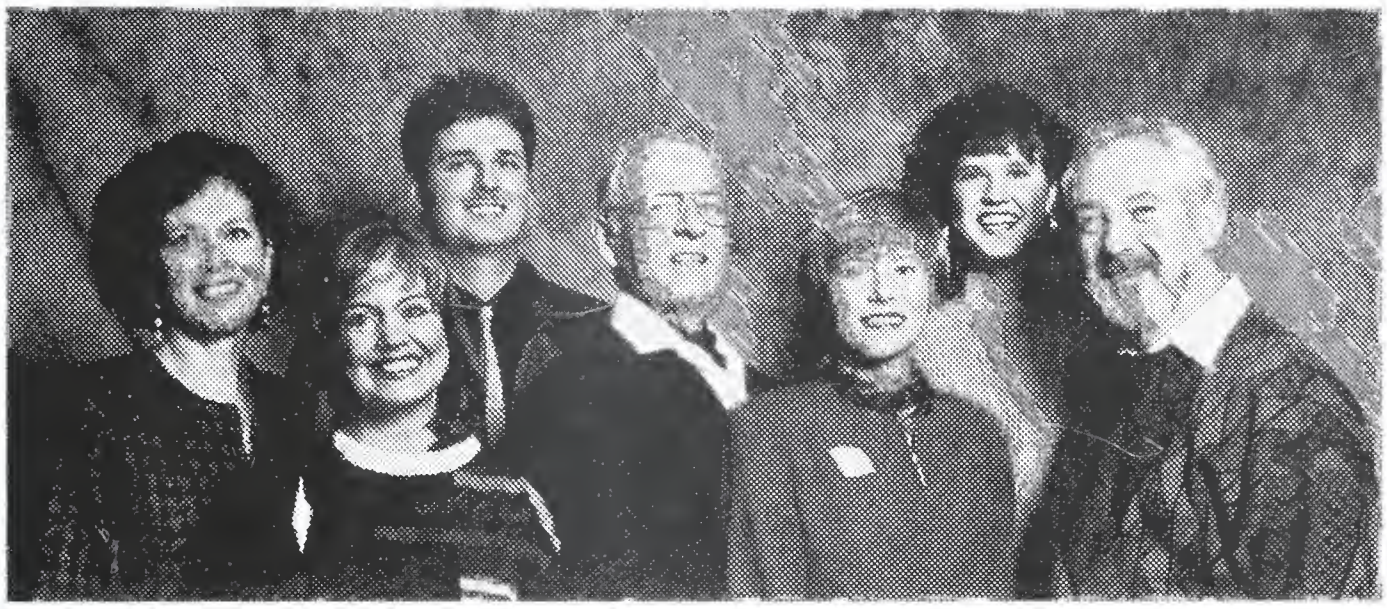

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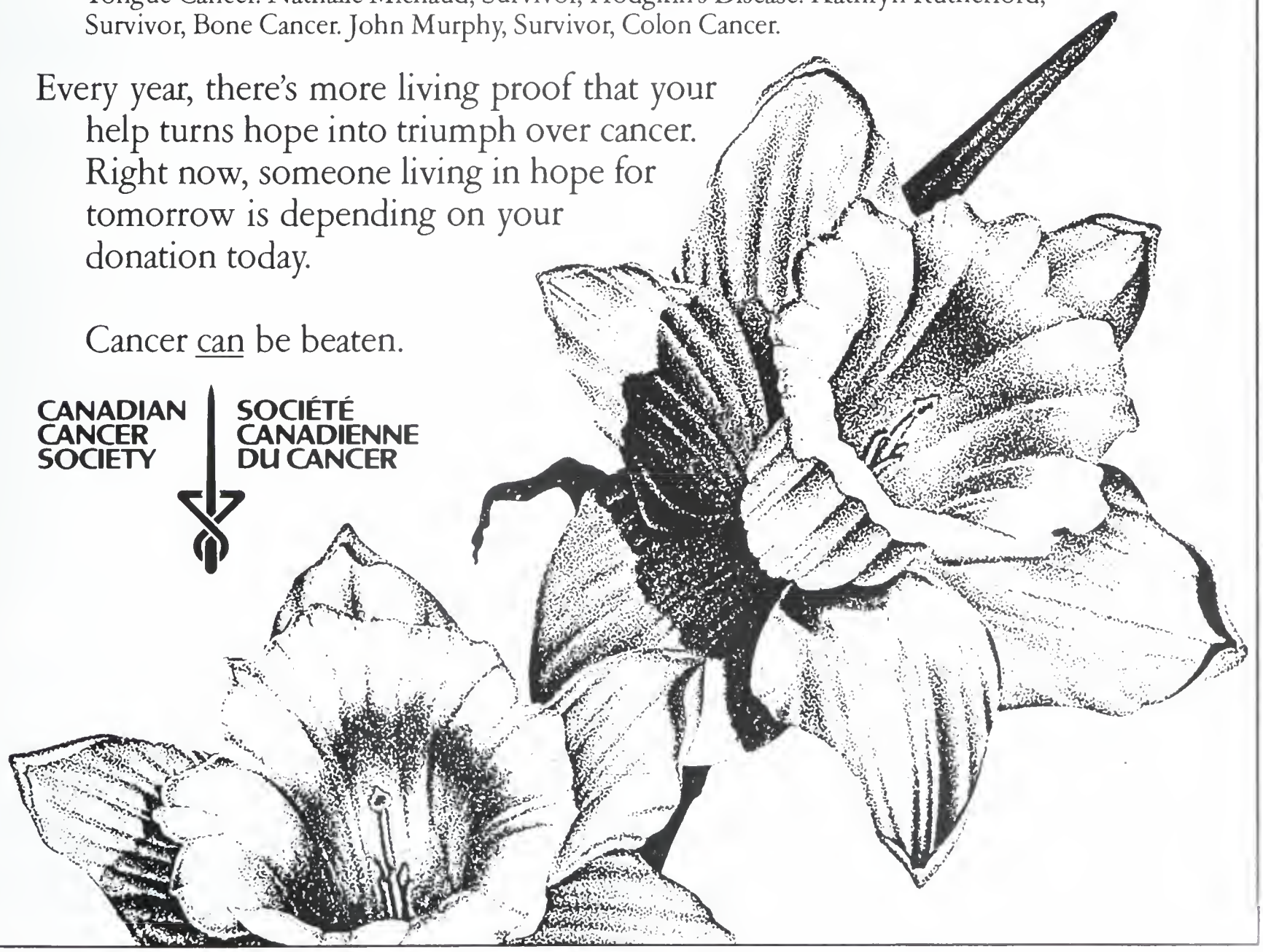

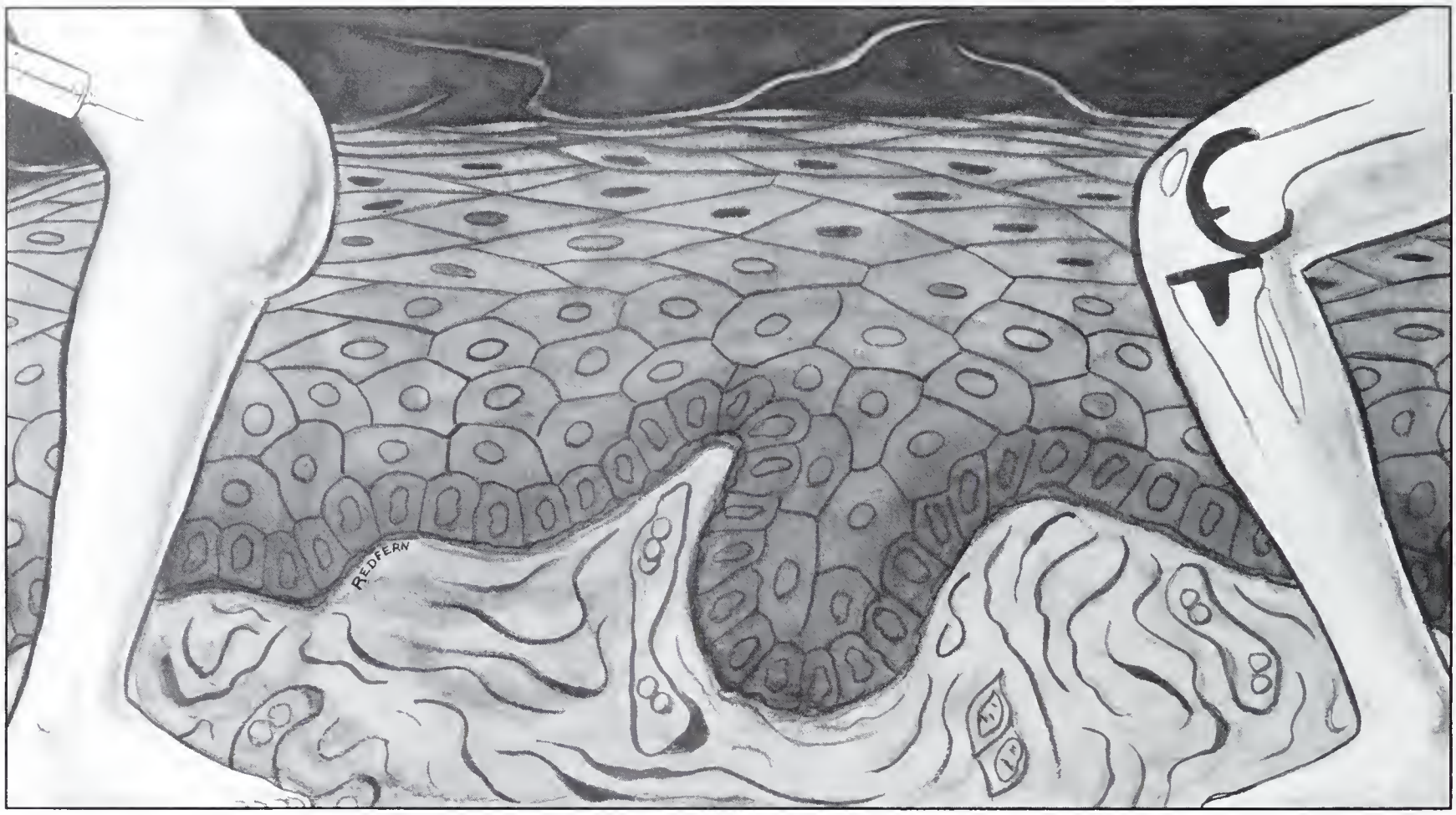

CHRISTINE REDFERN

The Use of Tissue Expanders in the Revision of a Knee Fusion to a Total Knee Arthroplasty

Philip.R. Solomon, B.A., 9T4, Lars-Uwe Lahoda, 9T3, Nizar Mahomed, M.D., Allan Gross, M.D., F.R.C.S.*

*Professor and Chairman, Division of Orthopedic Surgery, University of Toronto

Abstract

This article presents the first documented case where tissue expanders have been utilized in the conversion of a knee fusion to a total knee arthroplasty. The advantages tissue expanders provide to reconstructive orthopaedic surgery are reviewed and the potential complications of this technique are discussed.

Introduction

Tissue expansion is a natural physiological process which can be seen in lax abdominal and breast skin following pregnancy or massive weight loss. The same process is seen when skin, muscle and mucosa are stretched over underlying hematomas and tumours. The use of tissue expansion in a clinical setting was first attempted by Neuman in 1957 for the reconstruction of a congenital ear deformity[1]. It was not until 1976 when Radovan presented the procedure to the American Society of Plastic and Reconstructive Surgeons that tissue expansion gained acceptance as a useful surgical tool[2]. The technique involves forcefully injecting normal saline intermittently through a subcutaneous port into a dome shaped silicone implant over a period of a few weeks. As the expander enlarges, the surrounding tissue expands, providing tissue of the same colour, texture and thickness. Its applications have been primarily in the field of plastic surgery where it has been used to develop flaps in almost all areas of the body. To our knowledge, there have been no documented cases of the use of tissue expanders for the reversal of knee arthrodesis. We present a case where tissue expanders facilitated the conversion of a knee fusion to a total knee arthroplasty. The tissue expansion mobilized the scarred down quadriceps mechanism and also provided adequate skin and soft tissue for the procedure.

Case Report

A 41-year-old female wiring technician was seen in clinic for poor functioning of a right knee fusion which included difficulty in getting out of a car and getting in and out of theatre seats. Her knee problems began twenty-two years ago when she tore her medial collateral and anterior

cruciate ligaments. A year following the accident, an arthrotomy and a total medial meniscectomy were performed. Degenerative changes in the patello-femoral joint were noted. She became progressively more unstable and the following year reconstruction of her anterior cruciate ligament was performed. After six years an arthroscopy was done and revealed degenerative changes in her medial and patello-femoral compartments. Over the next two years she had numerous surgical procedures performed on her knee, including a high tibial osteotomy and open debridement. Seventeen years after her original injury, she had a second arthroscopy which was complicated by a septic arthritis with *Escherichia coli.* This was treated with fusion of the knee using an external fixator. The knee was fused in twenty degrees flexion and five degrees varus. The results were poorly tolerated by the patient for over two years and eventually the patient requested a conversion of the fusion to a total knee arthroplasty.

The patient was a strong candidate for a total knee replacement since she had good circulation, good quadriceps muscle strength, and adequate bone stock. However, the patient was noted to have tight skin and soft tissue around the quadriceps and there was concern that this could complicate the revision. Another concern was that the disuse atrophy and scarring of the quadriceps, which had occurred since the fusion, could seriously limit the range of motion of the knee following the arthroplasty. Tissue expanders were utilized to prepare the skin and soft tissue to facilitate the revision. A 386 cc tissue expander was placed under the quadriceps mechanism and a 191 cc expander was placed in the medial and distal end of the thigh (Fig. 1).

The tissue expanders were injected through subcutaneous ports once a week for a period of six weeks. This was followed by a successful conversion of a right knee fusion to an uncemented total knee arthroplasty (Fig. 2). Seven months postoperatively, the patient had active range of motion of five to ninety-five degrees and adequate ligament stability. At this time the patient was ambulatory, requiring no walking aids and was satisfied with the results.

Figure 1(a): A/P view of a right knee fusion. Tissue expander implants are seen at the distal end of the femur.

Figure 1(b): Lateral view of a right knee fusion. Tissue expanders are seen at the distal end of the femur. Long arrow points to 386 cc expander, short arrows outline 191 cc expander.

Figure 2(a): Post-operative A/P view of the right knee from the same patient as in Figure 1 following a total knee arthroplasty.

Figure 2(b): Post-operative lateral view of the right knee from the same patient as in Figure 1 following a total knee arthroplasty.

Discussion

Since Radovan's description of tissue expanders in 1976, the clinical applications of this technique have increased to include facial, neck, breast, and upper and lower extremity reconstruction[3]. The function of expanders has been to mechanically stretch tissue prior to surgery to provide excess tissue adjacent to the potential surgical defect which can be used then as a donor for reconstruc-

tion. While the exact mechanism by which the expanders work is not known, a number of current theories exist to help explain this phenomenon. Some believe that the forceful internal pressure created by the expander breaks molecular bonds and leads to sliding of collagen fibrils[3], whereas others postulate that it is the trauma induced by the expander which leads to the release of an enzyme that facilitates tissue expansion[3]. It is important to note that Radovan's observations showed that the donor flap maintains full thickness during expansion. The dermis appears thinner, but it is supported by an additional capsule layer and eventually recovers its full thickness[3]. Radovan also noted that as the flap expands it may show a bluish discolouration which he believed was due to ingrowth of new vessels within the expanded tissue[3].

It is important for surgeons using this technique to be familiar with all possible complications that can occur with its use. The reported problems include infection, necrosis of tissue leading to expander protrusion, and rupture of the expander[3, 4]. Local tissue damage can result from high internal pressures leading to ischemic damage as well as from hematoma occurring from implantation of the expander. Austad et al studied the effects of the pressure produced on local tissue[5]. Histological examination revealed that the pressure of this prosthesis did not cause local inflammation but did cause muscle atrophy.

The uses of tissue expansion in lower extremity surgery to date have included: removal of benign lesions, repair of contour defects, coverage of problem wounds, and mobilization of contracted skin and muscle[6, 7, 8]. Weinzweig documented the use of tissue expansion to facilitate a total knee replacement for a post traumatic arthritis[7]. In this case, tissue expansion provided the necessary skin and soft tissue for the procedure. Riederman described that the use of tissue expanders was useful in mobilizing the contracted tissue around the quadriceps, facilitating wound healing following a Maquet osteotomy[8].

In our case, the purpose of the tissue expanders was twofold. Firstly, the tissue expanders provided the adequate skin and soft tissue for closure of the wound following the total knee arthroplasty. Secondly, we believe the tissue expander elevated the scarred down quadriceps mechanism from the distal femur and, in turn, provided the patient with good post-operative range of motion.

Tissue expansion proved to be a helpful tool in the conversion of a knee fusion to a total knee arthroplasty where skin and soft tissue deficiency and impaired mobility of the quadriceps mechanism might have compromised the procedure.

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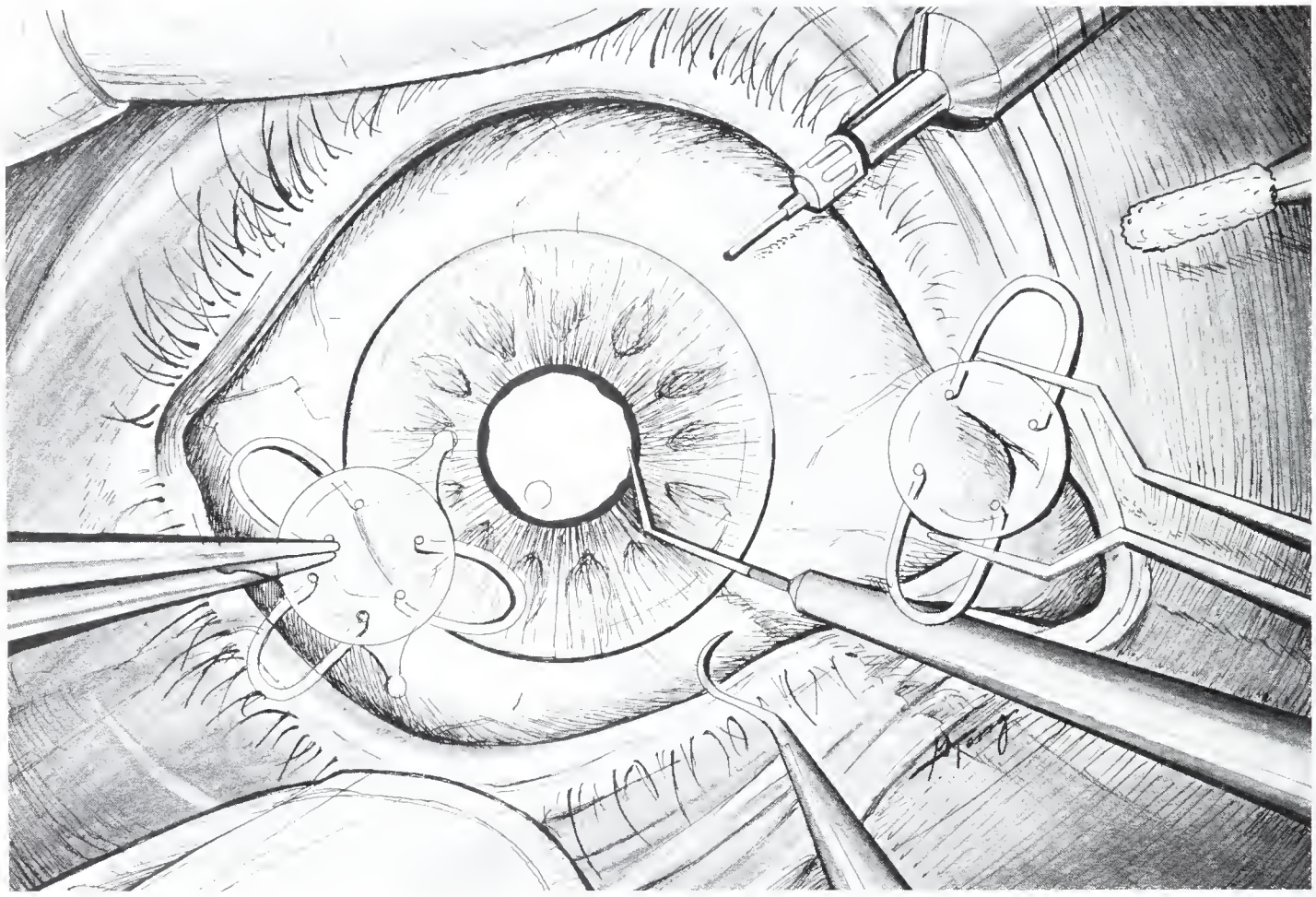

GLADYS TONG

Recent Trends in Cataract Surgery

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Abstract

Cataract surgery is one of the most common operations in patients over 65 years of age[1]. Although cataract surgery has been performed for more than fifty years, the technology of cataract extraction has progressed at an astonishing rate especially during the last two decades, and as a result various surgical approaches have evolved. The etiologies of different types of cataracts are presented and several surgical approaches are described and discussed in detail in terms of their merits, indications and possible complications with emphasis on the most recent advances.

Introduction

Cataract is the loss of transparency of the crystalline lens within the eye. Surgical lens extraction with the replacement of the optical power of the lens is the only effective method to restore vision. Nearly forty million people worldwide have cataracts requiring surgery with 80% of those in developing countries[2, 3]. In Canada, over forty-five thousand lens extractions are performed each year[4].

During the past fifty years, cataract surgery has undergone a profound evolution with the introduction of new surgical techniques made possible by developments in viscoelastic substances, microscopes, ultrasound equipment and intraocular lens implants. The majority of cataract surgery is now performed under local rather than general anesthesia and patients are usually discharged by the next day. For many years, intracapsular cataract extraction was the procedure of choice, but this necessitated aphakic spectacle correction which produced visual distortion and aniseikonia. By the early 1970's, refinements in extracapsular extraction allowed for the retention of the posterior capsule which reduced the incidence of complications and provided good support for intraocular lens implants. In the late 1980's, the desire for reduced astigmatism and earlier rehabilitation in vision led to growing interest in small incision phacoemulsification (ultrasonic fragmentation of the lens). Although phacoemulsification was

introduced in the 1960's by Kelman, surgeons were previously reluctant to perform this technique because of its technical difficulties as well as the lack of a powerful emulsification machine[5].

Cataracts

Although cataracts may be associated with a variety of conditions, the majority are associated with aging. Age related cataracts may have contributing causes as approximately 10% of these patients have diabetes mellitus while some have a history of prolonged glaucoma therapy or intraocular surgery. Prolonged exposure to ultraviolet light may also be a contributing factor. Congenital cataracts may be hereditary or due to prenatal exposure to drugs, infection or trauma. Juvenile cataracts may be caused by rubella, galactosemia, congenital syphilis, and diabetes mellitus. Cataracts may be secondary to trauma or systemic chemical agents. With perforating injury, proteins released from the lens may even stimulate antibody formation causing phacoanaphylaxis. Systemic disorders such as Wilson's disease, Fabry's disease, and hypoparathyroidism may also cause cataracts[2,6].

Cataract formation can occur in a variety of locations. In nuclear sclerosis, protein aggregates in the middle of the lens can scatter light rays to produce glare. Because there is hardening and sclerosis of the lens, older patients may experience an improvement in near vision as a result of myopia. In cortical cataract, slit lamp examination may reveal vacuoles, water clefts and spokelike opacities in the cortex. In posterior subcapsular cataract, fine granular opacities usually appear first in the visual axis of the posterior cortex and spread peripherally. Since opacities in the posterior zone of the lens are located in the path of the converging light rays, the visual impairment tends to be more significant than comparable opacities elsewhere. However, these patients may benefit from the use of mydriatic agents to ensure that more light reaches the lens.

In cataract formation, the stages of opacification are described as immature (incipient), mature, morgagnian (dissolution of cortical material), and hypermature. If a lens retains some transparency and vision is only mildly affected, then the cataract is termed immature, while an entirely opaque lens is termed mature. In some cases of mature cataracts, swelling of the lens may cause it to be closely related with the iris resulting in angle-closure glaucoma. Alternatively, in hypermature cataract, where the lens capsule becomes more permeable, the leakage of liquefied lens substance may stimulate an inflammatory response causing obstruction of the trabecular meshwork resulting in phacolytic glaucoma.

Indications for Surgery

Cataract extraction is usually indicated when the impairment of vision, even with the aid of eyeglasses or contact lenses, becomes unacceptable to the individual patient. Hence, this may depend on the patient's age, occupation, or hobbies. Occasionally, cataract surgery may be necessary if there is phacolytic glaucoma or phacoanaphylaxis present, or when visualization of the fundus is required to control diabetic retinopathy with laser therapy.

Operative procedures

Modern cataract surgery can be grouped into the following techniques: (1) intracapsular extraction (ICCE) with intraocular lens (IOL) implantation in the anterior chamber; (2) extracapsular extraction with IOL implantation in the posterior chamber; and (3) small-incision phacoemulsification. Local anesthetic is injected into the lateral lids or near the temporomandibular joint to block cranial nerve VII. A retrobulbar or peribulbar injection is also performed to block cranial nerves III, IV, V and VI, with II being variably affected.

Intracapsular Extraction (ICCE)

Intracapsular extraction, which is performed infrequently today, consists of removing the entire lens, including the capsule, with a cryoextractor. An enzyme is usually injected to lyse the zonular fibers in order to free the lens from its attachments. An iridectomy is performed to avoid adhesion of the iris to the vitreous surface which may lead to obstruction of the aqueous outflow from the posterior to anterior chamber.

Extracapsular extraction (ECCE)

This procedure can be divided into the following sequential steps: anterior capsulotomy (incision of lens capsule); expression of the nucleus; irrigation and aspiration of the lens cortex; cleaning of the posterior capsule; posterior chamber IOL implantation and wound closure. The surgical procedure begins with a superior rectus bridle suture to stabilize and position the eye in a downward rotation to facilitate surgical access to the limbus. The conjunctiva is incised and an ophthalmic cautery is used to provide hemostasis. After gaining access to the anterior chamber via a superior limbal incision, a viscoelastic substance (VES) is injected to maintain the anterior chamber. This serves to minimize corneal endothelial cell loss from trauma which can produce loss of corneal transparency[7,8]. A circular incision capsulotomy is then performed and the corneoscleral incision is enlarged to approximately 13 mm so that the lens nucleus can be expressed (Fig. 1).

Subsequently, part of the wound is closed but a large enough opening is left to insert a double-lumen needle for irrigation and aspiration of the cortical lens material. A volume of saline, chemically similar to the aqueous is injected to replace the volume of lens tissue removed. After the posterior capsule is cleaned, additional VES is injected to allow the capsular "bag" to open up during the insertion of an intraocular lens. Before the wound is closed, any remaining VES is removed and the anterior chamber is filled with saline. A subconjunctival injection of corticosteroids and antibiotics may be given before the eye is patched.

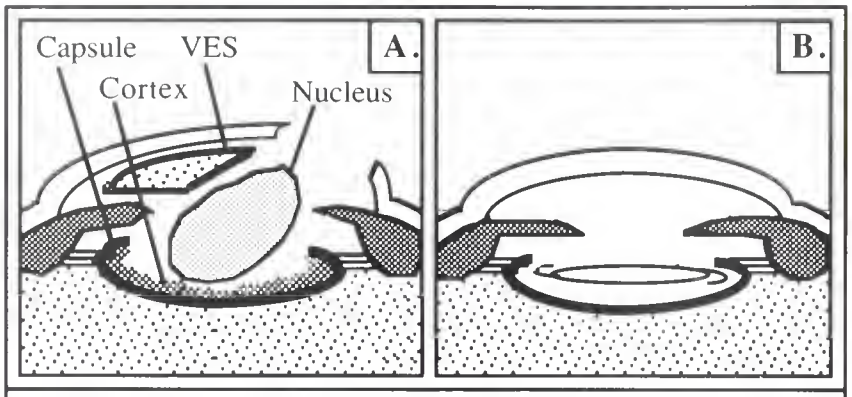


Figure 1: Extracapsular Extraction
A. Nucleus Expression and B. Posterior Chamber Implant
The nucleus is expressed after anterior capsulotomy and injection of VES. Cortical material is then aspirated and the posterior capsule is cleaned so a posterior chamber IOL implant can be inserted.

Phacoemulsification

Phacoemulsification involves fragmentation of the lens nucleus with ultrasonic vibrations and subsequent aspiration. The initial incision (3.5 mm) only needs to be large enough to introduce the tip of the ultrasound-powered double-lumen emulsification instrument. Ideally, a continuous tear capsulotomy is performed such that only a window in the anterior capsule is opened; thus, the structural rigidity of the capsular "bag" is maintained so that the lens nucleus can be held down in the bag during emulsification (Fig. 2A). Hydrodissection is then performed by injecting a balanced salt solution under the anterior capsule flap so that fluid flows underneath the nucleus and separates the nucleus from its cortical attachments. In some cases, another hydrodissection is performed to separate the soft peripheral cataract tissue from the hard central cataract tissue. The main purpose for hydrodissection is to allow rotation of the nucleus within the bag[9].

Different endocapsular phacoemulsification techniques may be applied to cataracts of different densities. Koch proposed that Spring surgery and Four quadrant cracking are reasonable methods for emulsification of soft and hard cataracts, respectively[9]. In Spring surgery, central sculpting and shaving of the nucleus with the phaco instrument is performed followed by a relaxing nucleotomy at the inferior nucleus (Fig. 2B). Since the phaco probe is inserted superiorly, the nucleus is then rotated 180° so that a relaxing nucleotomy can be performed on the opposite end more easily. Thus, two nuclear handles on each side are created so that a folding maneuver can be performed whereby the handles are pulled into the middle and emulsified[7] (Fig. 2C).

Given that denser cataract lenses may have difficulty "folding", a Four-Quadrant Cracking technique can be performed. This involves central sculpting and carving trenches perpendicular to each other by successively rotating the nucleus 90° (Fig. 3A)[9]. Because denser lenses tend to be more brittle, the phaco tip and a spatula can be

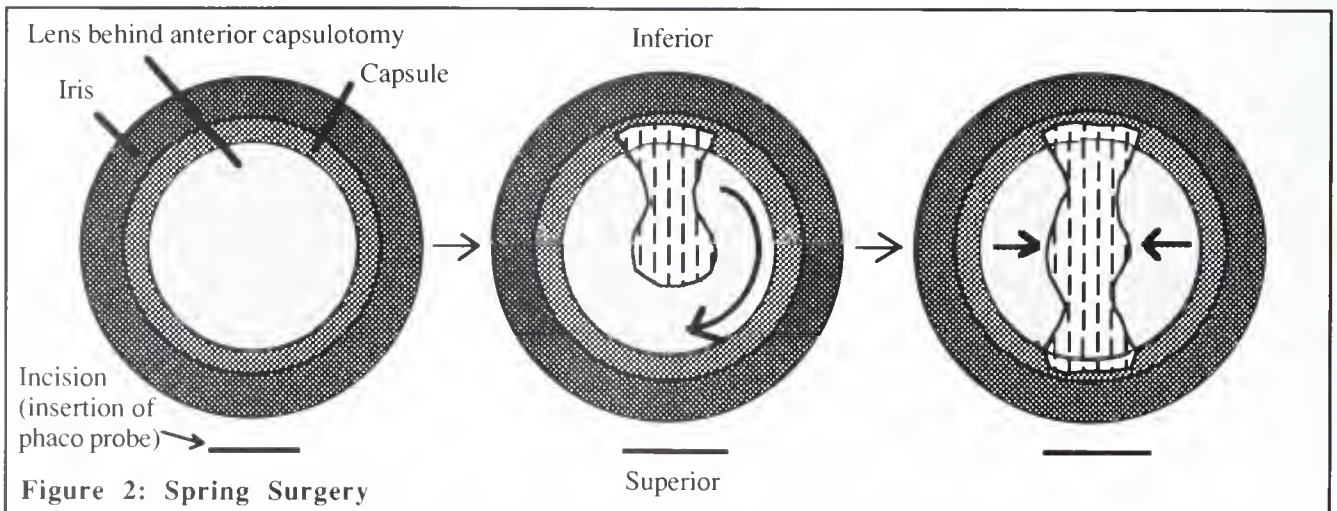


Figure 2: Spring Surgery

A. Anterior Capsulotomy and Hydrodissection are performed.

B. Central sculpting and a nucleotomy towards the periphery are performed with the phaco probe. The nucleus is then rotated 180° so that another nucleotomy can be performed on the opposite side.

C. A thin posterior plate is created to act as a hinge for folding (arrow) the opposite sides for aspiration and emulsification.

used to crack the nucleus into four quadrants (Fig. 3B). Each quadrant is then successively emulsified from the center to the periphery. Regardless of the technique used, the cortical material is aspirated in a similar manner to ECCE, though it is somewhat more difficult because the anterior capsulotomy is smaller. Given the small incision, greater abnormal curvatures in the cornea producing astigmatism. Because of these disadvantages, ICCE is rarely indicated except in chronic uveitis (see below) and subluxation of the lens where removal of the lens and its capsule is required. However, because of its speed, simplicity and lack of requirement for microsurgical instruments, ICCE is still used in mass intervention programs in developing countries[3].

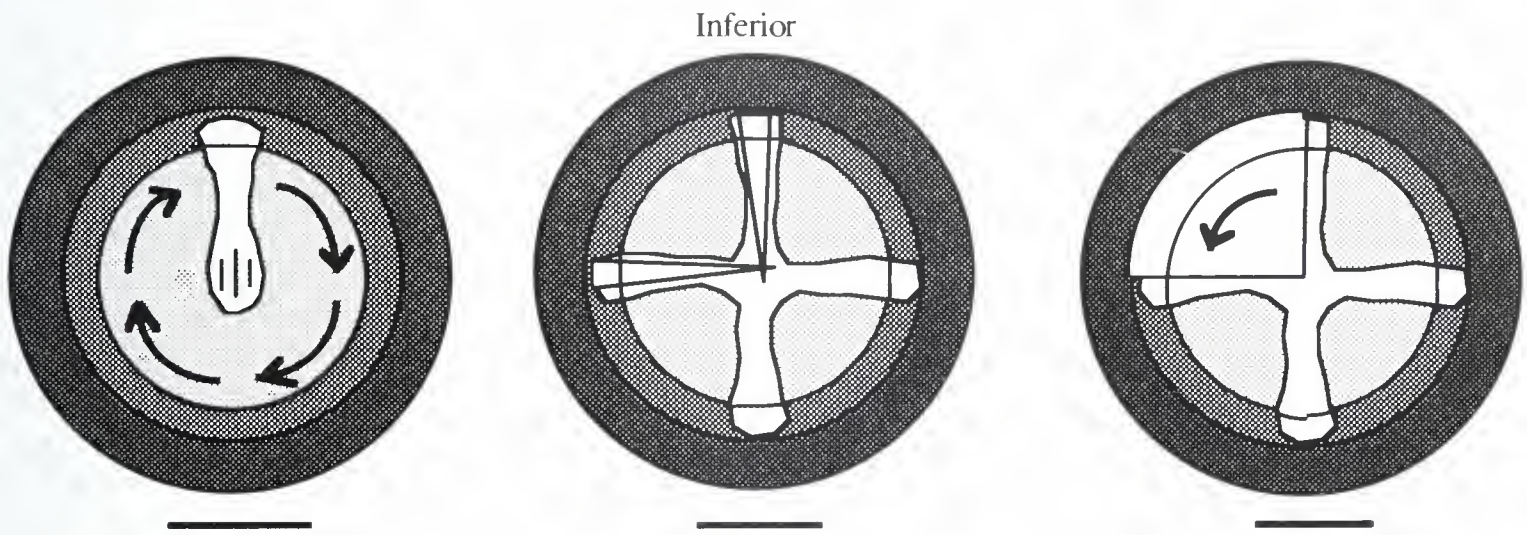


Figure 3: Four-Quadrant Cracking

A. After anterior capsulotomy and hydrodissection, (see fig. 2A) central sculpting as in spring surgery is performed but to a lesser degree. The nucleus is rotated 90° successively so that four "trenches" at right angles to each other are dug with a phaco probe.

B. The nucleus is then cracked open along the trenches. The apex of one quadrant is elevated slightly and emulsified.

C. After the first quadrant is emulsified, the nucleus is rotated counterclockwise so that the next quadrant can be cracked and emulsified and so on.

Fig.2,3: Modified from Koch, P. Converting to Phacoemulsification Slack Inc. 1992: p.73-75.

the surgeon can either insert a flexible small incision IOL or enlarge the incision for insertion of a posterior chamber implant.

Discussion

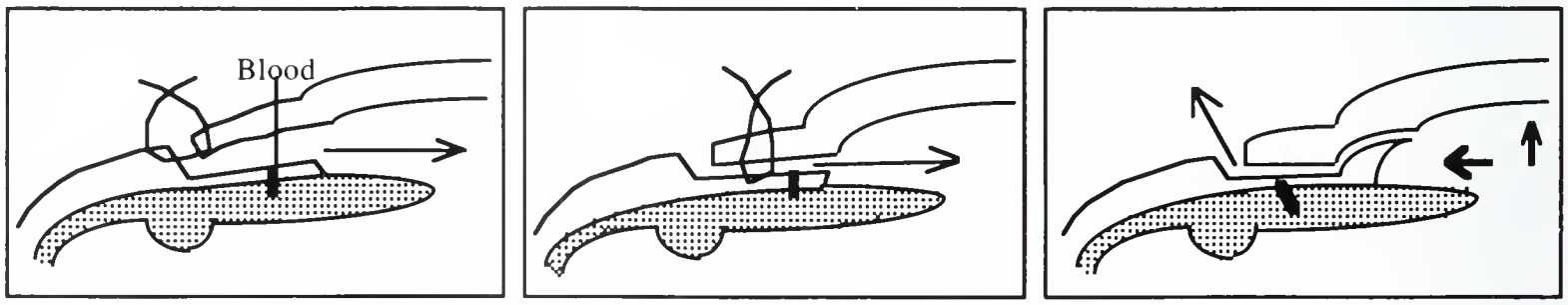
Intracapsular extraction (ICCE) was routinely used up to the 1970's. However, unlike ECCE where the posterior capsule is retained, in ICCE, the entire lens is removed. Hence, in ICCE there may be greater disturbances to the vitreous which account for its greater incidence of macular oedma and retinal detachment[7,12,13]. In general, the capsular bag is the ideal site for IOL implantation since it confers the greatest stability without contact with vascular tissue and is close to the nodal point of the eye[11,14]. Without the posterior capsule, ICCE patients are either given thick spectacles or an anterior chamber IOL implant, which may have long-term tolerance problems. In ICCE, the relatively large incision at the limbus necessitated by the removal of the entire lens typically results in

The majority of cataract surgery performed today in North America is ECCE. It is suitable for extraction of most types of cataracts. However, in patients with active chronic anterior uveitis where the posterior capsule can act as a scaffold for inflammatory membrane formation, ECCE is contraindicated. One complication of retaining the posterior capsule in ECCE and phacoemulsification is that opacification of the capsule occurs in 10-50% of patients within 3-5 years of ECCE or phacoemulsification[8]. Fortunately, discission of the posterior capsule can be accomplished with the neodymium: YAG laser noninvasively. However, this procedure should only be performed when visual loss is significant as it is associated with rare complications such as transient rise in intraocular pressure, damage to the intraocular lens, and retinal break or detachment[15,16].

Although ECCE represents an improvement over ICCE with respect to complications and the stability of the

implant, the smaller incision used in phacoemulsification provides additional benefits. Phacoemulsification provides greater anterior chamber maintenance due to a closed chamber, reduction in astigmatism, earlier improvement in vision and a stronger post-operative wound. Since smaller incisions have been associated with less inflammation, it is possible that the incidence of complications, such as intraocular hemorrhage and endophthalmitis may be reduced[17]. The incision in phacoemulsification is either closed with a single radial or horizontal suture or, if a corneal valve incision was made, no suture is needed (Fig. 4). Of the three methods, radial sutures provide the least control of astigmatism. Although horizontal sutures and corneal valve incision provide a physiologic closure of the incision and thereby control astigmatism, corneal valve incision results in a stronger postoperative globe, and a lower incidence of hyphema (blood in anterior chamber)[18,19]. Corneal valve incision appears to provide the best mean uncorrected visual acuity and visual recovery is usually within a week[20,21].

Figure 4: Incisions and Suture



A. Radial Suture
The cornea and scleral flap are pulled to a new position resulting in greater astigmatism. Blood in the sclera may track into the anterior chamber exacerbating hyphema.

B. Horizontal Suture
A more physiologic closure of the scleral tunnel confers greater control of astigmatism. However, blood can still track into the anterior chamber.

C. Corneal Valve Incision
The pressure in the eye (dark arrows) helps seal the wound so no suture is required. A more physiologic closure is achieved. Also, blood flow can no longer track into the anterior chamber.

Modified from: Koch, P.S. Structural analysis of Cataract incision construction. J of Cataract and Refractive Surgery. 1991. 17 suppl. : 661-7.

To fully realize the benefits of phacoemulsification, small incision IOL must be developed. The traditional polymethylmethacrylate (PMMA) lens with large optics (6.0-7.0 mm) and J-shaped haptics (ie. footplates) has proven to be safe and reliable. However, since PMMA is quite rigid, lenses with optics small enough to pass through small incisions (3-4 mm) may risk post-operative decentration. Since 1981, a great number of flexible or foldable lenses have been developed with silicone and iogel. However, their long term reliability has yet to be proven. Initially, some silicone lenses were associated with increased corneal endothelial cell damage and an increase in posterior capsule opacification[10,11]. However, this may be related to new insertional devices (e.g., folding forceps) and the surgeon's familiarity with their use. Another modification to modern posterior chamber IOL being assessed is the addition of UVR-absorbing molecules to the polymer of the lens since UVR below 400 nm can reach the retina after the cataractous lens is removed.

Phacoemulsification may be suitable for cataracts in children and in some elderly patients since emulsification of lenses that are soft or moderately hard tends to be easier. This technique may also be indicated in eyes that do not tolerate major surgery well such as those in congenital and traumatic cataracts. Moreover, the early recovery of visual acuity provided may be useful in children where stimulus deprivation amblyopia can develop quickly. However, consideration must be given to the fact that in very young infants, opacification of the posterior capsule develops quickly and the extremely soft nucleus does not separate easily from the surrounding cortex.

The disadvantages of phacoemulsification include the need for a highly skilled surgeon, highly sophisticated technology (e.g., ultrasound equipment) and a small incision implant lens. Moreover, depending on the surgeon's level of comfort, phaco-emulsification may be contraindicated in patients with small pupils or deep orbits restricting surgical access or corneal opacities which obscure the surgeon's view. Hard cataracts tend to be more to difficult to emulsify. Surgical trauma to the corneal endothelium, the posterior capsule, and iris is possible since the ultra-

sonic probe operates in close proximity. A rare but serious complication is the loss of nuclear fragments into the vitreous through a capsule break.

Conclusion

Various methods of cataract extraction aimed at improving postoperative visual acuity and reducing the incidence of complications have been developed over the past decades. This has been made possible by developments in surgical equipment and intraocular lens implants and the increase in understanding of wound architecture. Although ICCE is an economical and simple approach suitable for mass intervention programs, ECCE has proven to provide a more stable location for intraocular lens implantation and a lower incidence of complications. At the present, there is an increasing preference for small-incision phacoemulsification which provides the further advantages of greater astigmatism control and earlier rehabilitation. However, phacoemulsification is not without risks as it is technically more difficult and may not be suitable for all patients. Thus, in the selection of a surgical technique, the patient's age, type of cataract and the presence of underlying eye disorders, must be carefully considered.

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Reports from the University of Toronto's International Health Program

This summer, many of our students from the University of Toronto Medical School had the opportunity to go to a third world country and see for themselves how medicine is practised in a foreign land. All of the students who participated found it to be an enriching experience that broadened their minds as well as their medical knowledge. Those of us who were not able to go were fascinated by the stories and accounts that they brought home with them. As such, we felt is was appropriate that they share their stories with our readership. The following are the accounts of some of the experiences that Jeff McTavish and Alison Leggett encountered in Kwazulu, South Africa and West Borneo, Indonesia, respectively.

Zululand

Jeff McTavish, 9T3

Zululand. It is the only place on earth to which the once-mighty Zulu tribe is native. Far from Western influence, the tribe's lifestyles and traditions have remained unchanged for centuries. I had the opportunity to experience medicine in this fascinating culture, and it was an adventure that I will not soon forget. For 3 months, in the summer of 1991, I lived in a Zulu homeland within the province of Natal, South Africa.

My education in South African medicine began long before I reached Kwazulu. My first encounter occurred not in a private or public hospital, but far lower in the multi-tiered system. Freshly off the plane, I was invited to spend the evening at a clinic on the edge of Alexandra, one of Johannesburg's larger black townships. I was warned of the considerable violence that had occurred in Alexandra during the months prior to my arrival, including a shooting death in the clinic's entrance. Sure enough, my summer began with a first-hand look at the continuing effects of Apartheid.

The area surrounding Alexandra was astonishingly affluent and was scattered with restaurants and car dealerships. The contrasting squalor of the township, however, was equally surprising. In the space of 300 metres, the bright modern street was suddenly transformed into a gravel road bordered by rows of decrepit plywood shacks, lit only by scattered small fires. The vague human shapes that could be discerned in the fiery glow created a truly eerie atmosphere. As we turned into the clinic, barely 100 metres from the first huts, my South African companion explained that sane white civilians rarely entered Alexandra, and never after dark.

The clinic was small and closely-guarded. While primarily equipped for uncomplicated trauma, it employed a full-time ambulance to transport more serious injuries to a larger centre. Thus, high quality medical care was theoretically available to the clinic's patients. In the larger scheme of things, however, patients from the townships were considered low priority, and had limited recourse when treatment was poor. Once inside, I found that the clinic was entirely staffed by medical students, who singlehandedly treated severe burns, lacerations and frac-

tures. Within hours of my arrival, we had seen two basal skull fractures, two fractures of the frontal sinuses and many lacerations of the face and scalp. Most injuries were the result of alcohol-induced disputes settled with bricks and knives, but several could be attributed to seemingly random attacks by the South African Police. On a number of occasions police tanks could be seen rumbling into the township to "keep the peace". I was told that the police and army were part of the few remaining collectives of white elitism, perpetuating racism, while much of the population supported the movement toward black equality.

As Johannesberg was merely a pit-stop on my journey, it was soon time to move on. The 20-hour bus ride, deep into Kwazulu, was like a trip back in time. The hospital where I spent most of my time was nestled in the Lebombo mountain range, within sight of Swaziland and 30 kilometres south of Mozambique. By daylight, the area was breathtakingly beautiful with a view for kilometres. The barely discernible Zulu homesteads were tightly structured circles of thatched huts built around a central cattle corral. I discovered that the Zulus took great care to isolate their homes from their neighbors out of a fear of witchcraft accusations. In the Zulu society, such accusations could arise from even harmless situations such as straying animals or children. Prior to my arrival in Kwazulu, a witch hunt took place in nearby Kwangwanase, resulting in a series of murders and beatings.

The Mosvold Hospital began as a mission station in 1911, but was taken over in 1980 by the Kwazulu government. Its staff consisted of five full-time physicians from across the globe, none of whom planned on spending more than 3 years in such an isolated area. However, the true driving forces within the hospital were the African nurses and midwives who ran the wards and performed many vital community functions.

The hospital was short-staffed, so I began to see and treat patients on my own almost immediately. The injuries I encountered were as different as the culture itself. My early patients included a 73-year-old woman who had broken her hip in a fall from a tree. Despite our pleas, she would not promise to give up tree climbing once she recovered. More sobering, perhaps, was the case of three young girls who were seriously injured when they set off a mine near the Mozambiquan border. My responsibilities increased quickly, and by summer's end I was performing simple operations (e.g., tubal ligations) on my own.

Because it was a bumpy six-hour drive to better facilities, many difficult operations were performed at Mosvold. When such unusual operations were required, an interesting sight would occur. The doctors, trained only as general practitioners, would complete these difficult tasks with the step-by-step assistance of an open surgical text. Operations took on a festive air with continuous singing by both nurses and patients throughout the procedures.

A discussion of health-care in Zululand would not be complete without mention of the witchdoctor. Revered by Zulu society, this individual was always consulted (for a considerable fee) before western medicine. A history and physical examination were rarely necessary, as throwing bones and beads was the primary diagnostic tool. The witchdoctors' high stature was largely unwarranted because the delay in proper treatment usually resulted in the patient's condition worsening. Furthermore, the treatment itself (most often a herbal enema) commonly caused injury. How were these influential people selected? One criteria was the content of a Zulu's hallucinations at a young age (an experience rarely noted on Canadian medical school applications).

The biggest single barrier to the practice of medicine in Zululand was an almost nonexistent understanding of the Zulu culture. The hospital compound was physically removed from the community, and medicine was prac-

ticed with little communication with the outside population. In addition, the situation in South Africa was somewhat unique, as Apartheid had legally prevented whites from living with blacks. This law was enforced by the army and, thus, there was little known first-hand experience of Zulu lifestyle.

The people at Ingwavuma were extremely friendly and welcomed outsiders immediately. Conversation was a true form of entertainment that knew no age boundaries. A bus-load of strangers would invariably be involved in animated conversation and laughter, giving an outsider the impression that they were all old friends. It was a refreshing change from the uncertainty and suspicion directed towards strangers in our cities. As well, I was consistently intrigued by the long conversations held between the young and the elderly. There seemed to be a mutual interest that went beyond simply respect for elders. They did not appear to have any concept of a "generation gap".

The Zulus' also shared a different sense of time. The relaxed and unhurried lifestyle would allow a several hour wait for a doctor, or at the edge of a dirt road for a ride, without any hint of irritation. A popular source of humour was a foreigner (usually only missionaries visited) who foolishly expected pre-arranged times or appointments to be met. As it were, nothing in Zululand ever happened on time.

Unfortunately, there were also many harmful traditions maintained by the Zulus. In this male-dominated society, women had few rights. They had little choice in their mates, and were treated as possessions in marriage. Wives were not even allowed to eat with their husbands, but were instead forced to eat with the children.

Child abuse was also common, and took many forms. Father-daughter incest was constantly confirmed by cases of prepubescent girls with venereal disease. Also, a strong social stigma against twins often led mothers to neglect one twin to the point of death. However, as disturbing as these situations were, doctors often felt they had no right to intervene in a culture they did not understand, and simply treated the damage.

Despite the many difficulties that exist in Zulu society, the people had much to offer, and most of my memories are good. I remember a happy, outgoing community where every single person on the road would wave. Life was refreshingly simple and, for me, every day was an adventure. I learned to appreciate the beauty of the Zulu culture while also gaining an understanding of the problems that existed. Further, it has allowed me to view Canadian health and health-care in a different perspective. I am sure that this knowledge will shape the way that I practice medicine in the future.

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A Refuge in the Jungle

Alison Leggett, B.Sc., 9T4

The view from the four person Mission Aviation Fellowship plane as I flew over West Borneo was of a rough jungle on either side of the large Kapuas River, as far as the eye could see. I had expected rice fields but these disappeared soon after leaving Pontianak. It was hard to believe I had been in the bustling high-tech city of Singapore only one and a half hours earlier. Everything was such a lush green.

Tucked in between the hills, seemingly a million miles from civilization, a patch of buildings and a long empty field came into view. As we approached, that empty field became an airstrip and the buildings became a hospital complex. I had arrived at Bethesda Hospital.

My first impression of the 2 km area around the hospital was analogous to an oasis in a desert. I soon learned that this impression was not far from the truth. The health care delivered at Bethesda was literally life-saving and so needed that Indonesians and Westerners came from all over Kalimantan to be seen.

Monday and Wednesday were clinic days. The eight doctors (two American and six Indonesian) began their day making rounds by visiting the 200 in-patients. They then went to the clinic to see approximately 200 outpatients. Although Tuesday and Friday were surgery days, an equal amount of further surgery was performed during evenings and on non-surgery days. The clientele consisted of the very rich, who came to be seen by a Western doctor, and the very poor, who came because they knew they would not be turned away. The poor would receive the same care, but only pay what they could afford.

The range of medical pathology seen at Bethesda was quite incredible. Patients with twelve kilogram fibroids and grape-fruit size thyroid tumors were routine admissions. However, what didn't become routine were the people and the stories behind them. I remember meeting a frail, elderly woman in clinic one day for back pain. She probably did not weigh more than 40 kg. She told me the back pain was getting worse because "I used to be able to carry 50 kg of rice in my basket to market and now I can only carry 40". I thought to myself, young and healthy as I am, I couldn't carry 40 kg of rice over the thin trails that these women did.

Serious trauma, mostly from motor vehicle accidents, was a part of daily life and as a result the O.R. rooms were busy day and night. A few days after I arrived, a 19-year-old was brought in a wheelchair by his family to see what we could do to help him. Four months earlier he had been in an accident and was taken to the closest government hospital. He had a fractured femur, radius and ulnar bone. The fractures were bad but the worst was yet to come. In the government hospital, the doctors did not set the radial fracture and put the wrong pins in both his ulnar and femur. He also developed osteomyelitis in the femur and a compartmental syndrome in the forearm and leg, all of which went untreated. After having spent two months in the government hospital, he left with such extensive nerve damage that he had no movement in either the arm or leg. I was asked by one of the Indonesian doctors if I would like to see his X-ray and I realized that I was being asked to do an "orthopedic consult". The Indonesian doctor, with four years of surgical residency training, actually expected that I would have something valuable to contribute to the management of this case. I tried my best! We refractured and reset the femur but, needless to say, the boy will never walk again.

There were daily cultural hurdles to be maneuvered when working with the Indonesian doctors. They assumed that I would know so much more since I was educated in the west. I was flattered by their confidence in me, but often felt completely inadequate for the task. The following is one example of the communication and cultural barriers. During an extremely busy two-week period, the staff consisted of only three doctors and myself and we were overrun with patients. We had a young brain-dead patient using the only ventilator and oxygen supply. Meanwhile, an otherwise healthy woman with COPD proceeded to go into respiratory failure. I strongly suggested that the oxygen would be put to better use on the woman in respiratory failure but the doctor in charge was not comfortable with that idea. I looked in on the woman later that day and her clothes were drenched with sweat from fatigue. Trying not to "step on the doctor's toes", I again strongly suggested we intubate her and take the brain dead patient off the ventilator and the doctor finally agreed. I began bagging the woman manually, but the physician returned from speaking with he brain-dead patient's family and stated he couldn't bring himself to remove the respirator. He explained that the brain-dead patient had it first and it was therefore his right to continue using it. We were now stuck. The choice was to either bag the woman manually all night, or extubate her. In the end, the brain-dead patient continued to use the ventilator and, after several hours, we extubated the woman with COPD. She died the next day. There were many situations where patients died and we could not help them, but a patient dying as needlessly as this was incredibly frustrating. It would not be my last frustration during my stay.

My summer experience would not have been complete without trips to the villages farther interior. It was a one

hour flight on a plane packed with medications and supplies for our five day stay. We flew to villages that were completely inaccessible by land or water. I was one of the two white-skinned members of the five member team, and the only "doctor". The fact that I was now able to communicate reasonably well in Indonesian helped significantly. We arrived in one village at night because the trail to it, which we had expected to be easy, involved crossing swinging rope-bridges and wading waist-deep in cold rivers. I glanced down at the ground as we walked to the chief's hut and realized there was not a stone or twig to be found. The dirt road, I was told later, had been swept for hours in anticipation of our arrival and it looked like a royal carpet. I had still not seen any villagers by the time I leaned against the door to the chief's hut to take off my shoes. When I looked up, standing less than one meter away in a circle around me, were 70 scantily clad children watching my every move. I doubt that I will ever see eyes that wide again. I had thought that by this time I was used to being stared at because of my white skin. However, this welcoming committee was quite overwhelming and the crowd of children became my shadow for the next two days.

Despite the differences between us, I learned that people are very much the same. In these far forgotten villages of Indonesia, the children laugh and explore the world around them just as children do here in Canada. They experience grief and pain just as intensely as we do and people search for a deeper meaning and purpose beyond life on this earth. I am grateful for the opportunity that I had this past summer. I am confident that I will not forget the lessons I learned or the people behind those lessons because I know that a little bit of myself was left behind at the place I call "the refuge in the jungle".

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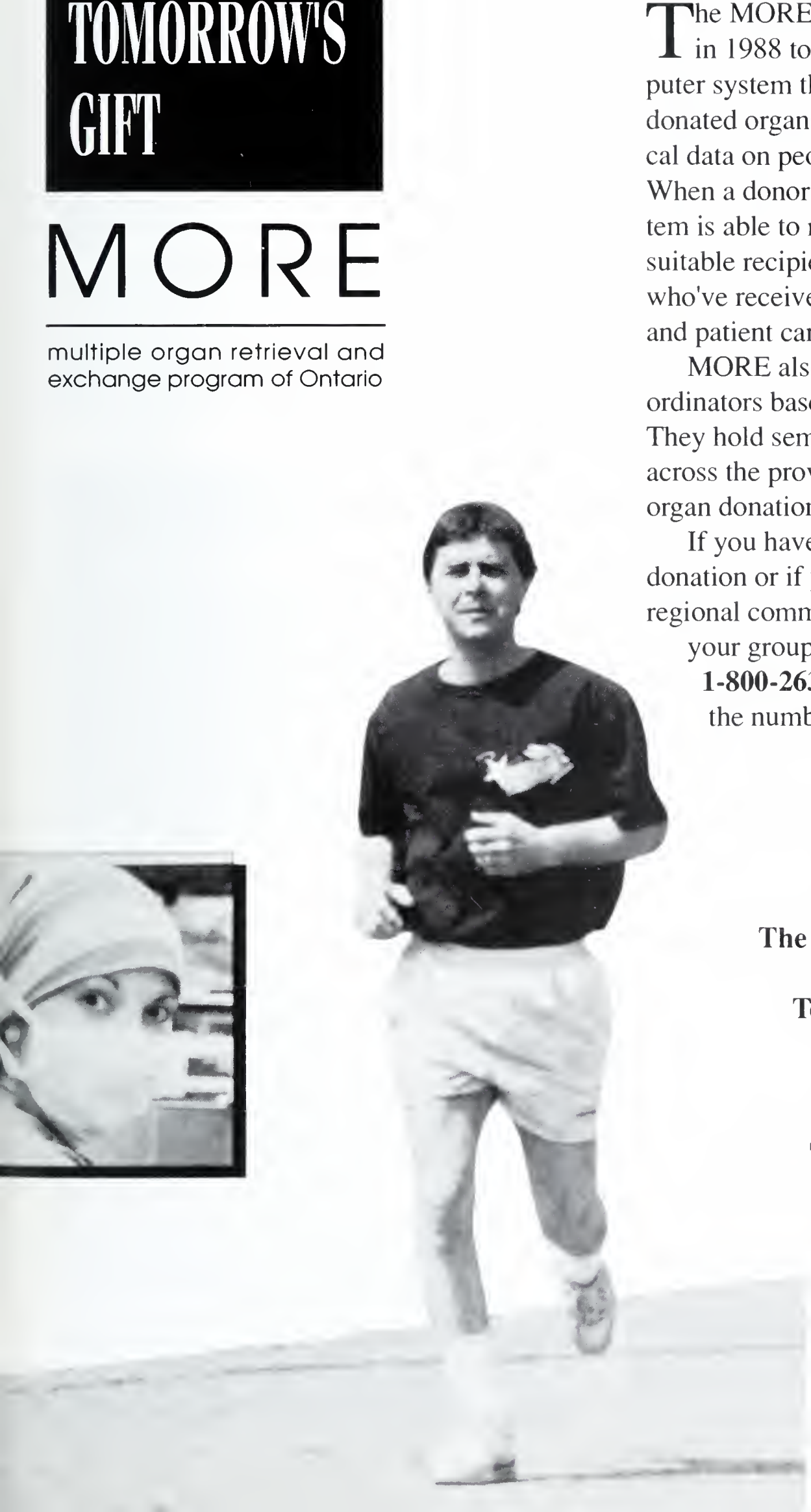

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Paediatric Presentations

Janine A. Flanagan, H. B. Arts Sc.

Special acknowledgment to Patricia Parkin, MD, FRCP(C), at The Hospital for Sick Children for the case illustrations and comments.

Case 1

This one-day-old neonate was born at term at Mount Sinai Hospital by C-section. The Apgar scores were recorded as 8 and 9. On examination of the infant, the most startling finding was some poorly organized spinal cord tissue covered by a pinkish, delicate, semi-transparent membrane on the posterior surface of the lower back at midline. The infant's lower limbs were held in flexion at the hip, with extension of the knees and equinovarus positioning of the feet. There was poor active movement of the lower extremities. The patient also had an enlarged head with a prominent anterior fontanelle.

What is the lesion, and what level is the lesion?

Answer to Case 1: This infant has a meningomyelocele, not a meningocele; the difference being that the former herniated sac filled with spinal fluid contains not only meninges (dura and arachnoid), but additional neural elements. One would also expect normal motor leg function with a meningocele unless other dysplastic changes of the spinal cord were present.

Of the defects termed spina bifida cystica, 75% are meningomyeloceles and 25% are meningoceles. The incidence of meningomyeloceles is 2/1000, making it one of the most common congenital anomalies.

In this particular case, the lesion was lower lumbar with sparing of function down to L4. Most defects are lumbar or lumbosacral (70%). As a result, there was sustained contraction of the hip flexors, quadriceps, and tibialis anterior, but paralysis of the hamstrings, gastrocnemius and intrinsic muscles of the feet. In addition, the loss of S2-S4 would also cause neurogenic bladder dysfunction.

In 95% of lumbar or lumbosacral meningomyeloceles, the brain demonstrates a Chiari II malformation: caudal displacement of the cerebellar vermis, pons, and medulla through the foramen magnum. This blocks the flow of CSF and causes hydrocephalus.

It is generally understood that an undetermined insult that prevents normal fusion and closure also causes malformation of cellular proliferation, migration and architectonics.

Surgery is recommended before the first week of age, with closure of the lumbar lesion, and placement of a ventriculo-peritoneal shunt if hydrocephalus develops. The talipes equinovarus will require corrective casts to correct positioning of the feet. More severe lower limb deformities will require surgery. The child will be started on intermittent bladder catheterization to ensure that the neurogenic bladder is adequately emptied. Urine cultures need to be monitored for the development of urinary tract infections, and the child may be placed on prophylactic antibiotics. The child will also need to be followed by a physiotherapist.

Research at present suggests that preconceptual folic acid will prevent the development of a neural tube defect. Such treatment is being considered in women who have had a previously affected child.

Figure 1: herniated sac

Figure 2: clubbed foot

Case 2

An otherwise healthy 4-year-old girl presented with a subcutaneous mass in the orbital area. The parents claimed a small mass had been present there for some time, but had grown to its present 1 1/2 cm size within the last week or so. There was no pain or tenderness associated with the mass, but the parents were concerned that it may obstruct vision if it continued to grow at such a rate. They questioned its removal, nonetheless, for cosmetic reasons.

Physical examination revealed the mass as firm, nontender, and highly mobile. She was also noted to have several cream-brown macular, cutaneous lesions, with a predilection for trunk and extremities. She also had some axillary freckling/hyperpigmentation. Her past medical history included a one-time seizure-like episode.

What is the preliminary diagnosis, and what evaluation would you undertake?

Answer to Case 2: The child appears to have Neurofibromatosis [NF-1] (Von Recklinghausen's Disease), a genetic disorder with autosomal dominant inheritance found in 1/3000 births, although there is a 50% new mutation rate.

There are two forms of NF. NF-1 is associated with chromosome 17; while NF-2 is associated with chromosome 22, bilateral acoustic neuromas, and is much less prevalent.

According to the National Institute of Health Consensus Development Conference[1], in order to diagnose NF-1, two of the following criteria must be present:

1. > 6 cafe-au-lait spots > 5 mm greatest diameter in prepubertal children, or > 15 mm in postpubertal children
2. axillary or inguinal freckling consisting of multiple hyperpigmented areas of 2-3 mm in diameter
3. 2 or more hamartomas (Lisch nodules) in the iris
4. 2 or more neurofibromas or one plexiform neurofibroma
5. osseous lesion such as sphenoid dysplasia, kyphoscoliosis, or pseudoarthroses
6. optic glioma
7. a first degree relative with NF-1 according to the preceding criteria

In this case, the patient fulfills criteria 1,2, 4, and 6. NF-1 is the consequence of an abnormality of neural crest differentiation and migration during the early stages of embryogenesis, possibly related to the influence of nerve or glial growth factor. Of the clinical manifestations mentioned, some are congenital, while others arise gradually in childhood and throughout adult life.

Unfortunately, there is no specific treatment for NF-1. Genetic counselling and early detection of treatable conditions or complications are priorities. Occasionally, neurofibromas can become symptomatic when they entrap a nerve root at the intervertebral foramina. Other complications include an increased incidence of malignant neoplasms and seizure disorders. In addition, mental retardation, renal artery dysplasia causing hypertension, stenosis of the aqueduct of Sylvius leading to obstructive hydrocephalus, and pheochromocytomas can be found.

Current recommendations for the evaluation of NF-1 include baseline CT scan or MRI of the brain and optic nerves to rule out optic glioma, and an ophthalmologic examination. Asymptomatic patients should be reexamined annually with a neurological assessment, including BP, auditory and visual screening, scoliosis screening, and a search for complications.

References

1. Listernick R., Charrow J. Neurofibromatosis type 1 in childhood. The Journal of Pediatrics 1990;116(6):845-853.

Figure 1: skin hyperpigmentation

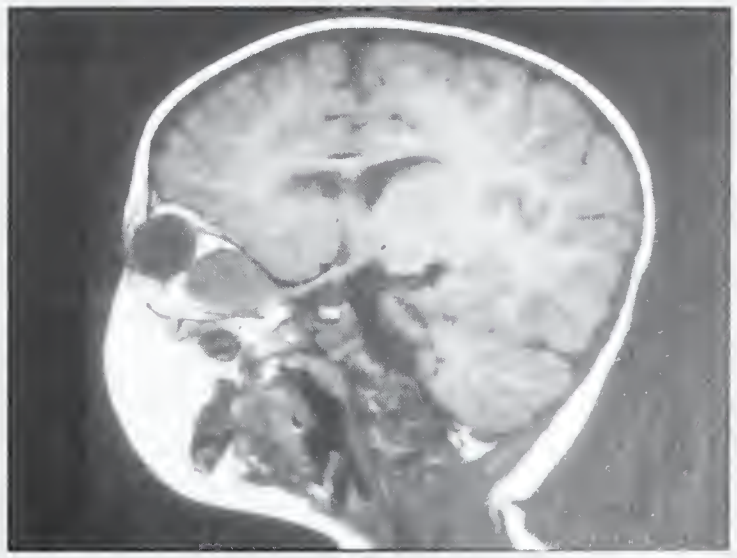
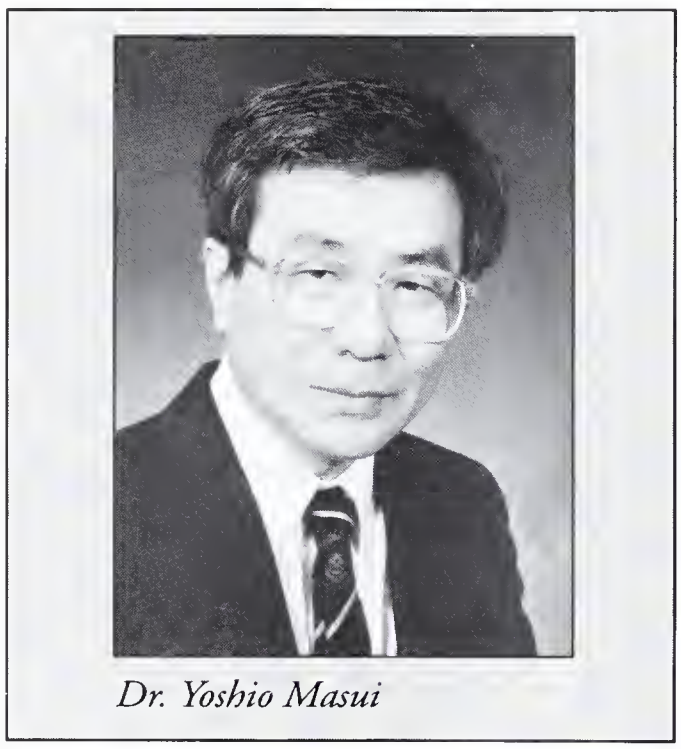


Figure 2: M_1W (600/20) oblique MRI

The 1992 Gairdner Foundation International Awards

D'Arcy Little, 9T5

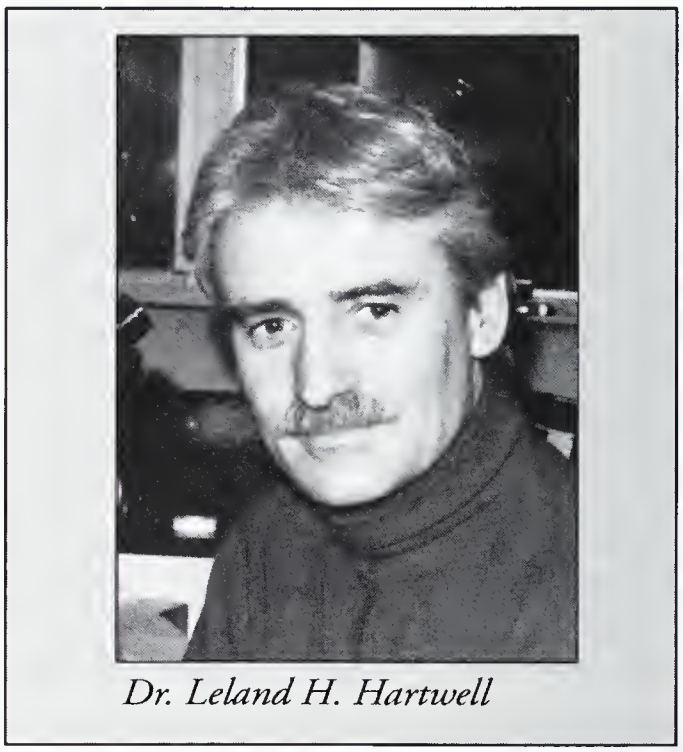
On Friday, October 23, seven scientists received the 1992 Gairdner Foundation International Award for achievements in medical science. Of the seven winners to receive awards totalling $220,000 (Can.), two were from Canada, two from England, and three from the United States. The recipients for this year were selected by the Gairdner Foundation's Medical Advisory Board, chaired by president, Dr. Charles Hollenberg. The awards were presented by The Honourable Henry N.R. Jackman, Lieutenant Governor of Ontario.



Dr. Yoshio Masui

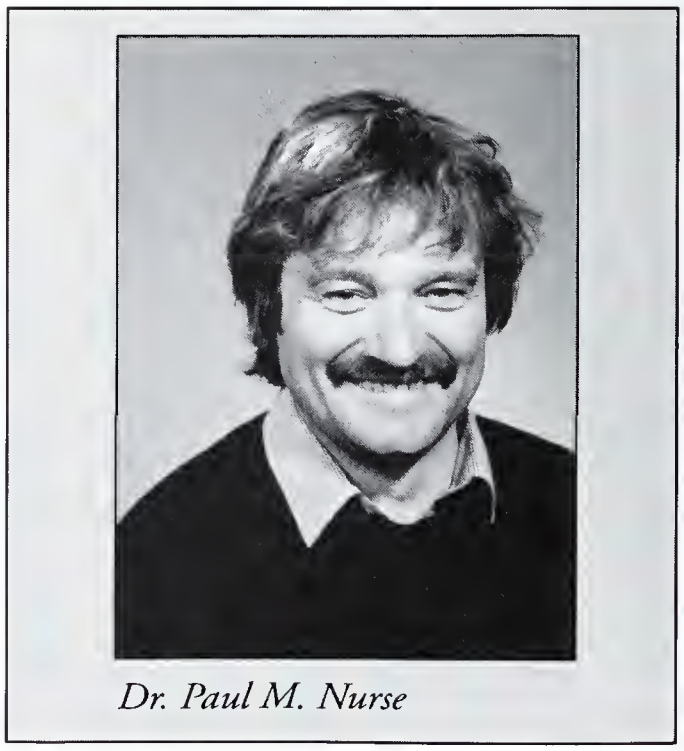
Three of the recipients, Dr. Leland H. Hartwell of the University of Washington, Dr. Yoshio Masui of the University of Toronto, and Dr. Paul M. Nurse of the University of Oxford were recognized for contributions in the field of cell cycle regulation. As most will remember from grade school biology, all living matter is made up of cells, and every existing cell has arisen from a previous one. Thus, growth, development and even life itself depends on the ability of a cell to divide and produce copies of itself. The process of cell division involves several complex phases which sequentially make up what is called the "cell cycle".

In the early 1970's, Dr. Masui, using biochemical methods, discovered maturation promotion factor (MPF) and cytostatic factor (CSF), two factors which play major and universal roles in the regulation of cell division. Dr. Masui also developed assay methods for these factors.



Dr. Leland H. Hartwell

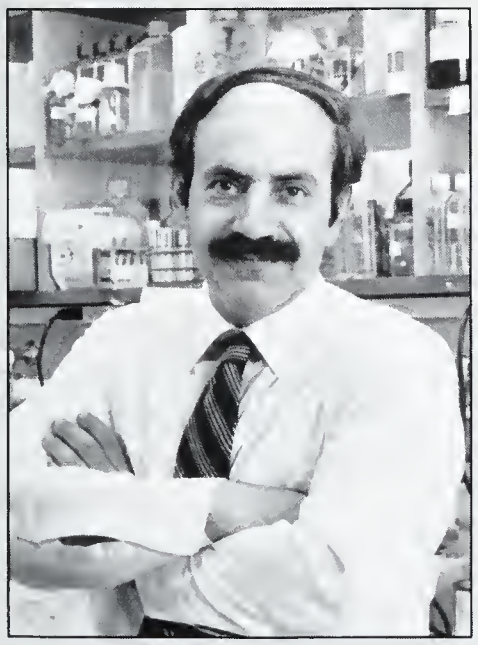
At approximately the same time, Drs. Hartwell and Nurse, working independently on different strains of yeast, and using genetic techniques, isolated mutants blocked at various stages of cell division. These mutants allowed the scientists to pin-point various genes controlling the cell cycle. More recently, it has been realized that one gene isolated by Dr. Nurse (cdc2) is the same as one of Dr. Hartwell's genes (CDC28), and that this gene encodes a protein equivalent to a component of Dr. Masui's MPF. The important work of these three scientists in elucidating the basic mechanisms regulating cell division set the stage for subsequent discoveries of cell cycle regulators by many other scientists.



Dr. Paul M. Nurse

Dr. Bert Vogelstein

Two of the other recipients, Dr. Bert Vogelstein, and Dr. Robert A. Weinberg, were honoured for elucidating the genetic events leading to the development of specific cancers. Cancer, a major cause of death, is a disease that appears to occur when cells in a particular tissue in the body begin to divide rapidly and without control. The mechanism of conversion of a normal to a cancerous cell involves a series of mutations which convert normal genes in the cell, called "proto-oncogenes", to "cancer-causing" genes called "oncogenes". This is the case, because it is the job of the proto-oncogene to control normal cellular growth . . . something it can't do after mutation.



Dr. Robert A. Weinberg

Dr. Weinberg made the discovery that the gene associated with the childhood eye cancer, retinoblastoma, acts not by causing abnormal growth, but by suppressing growth. Thus, as long as these so-called "oncogene-suppressor-genes" or "recessive oncogenes" are active, cancer is suppressed. Only when the recessive oncogenes are altered does cancer develop.

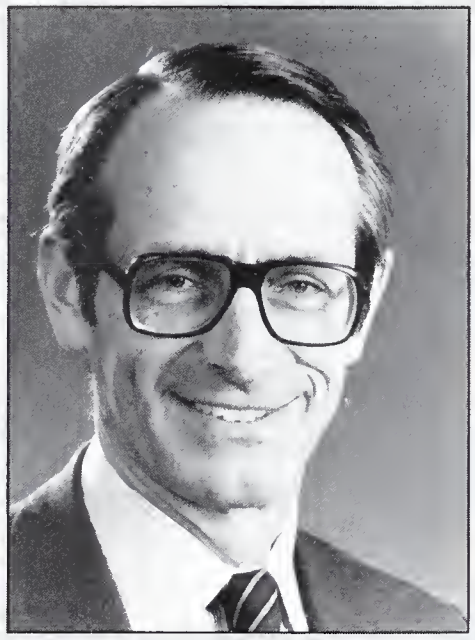
In a similar way, Dr. Vogelstein showed that colorectal cancer, a major cause of death in both men and women, is associated with a series of at least five genetic events involving disruption of several recessive oncogenes on human chromosomes 5, 17, and 18.

Professor Richard Peto was also honoured for his contribution to the design and analysis of clinical trials. Professor Peto, the world's leading statistician in the assessment of new therapies, has developed and

Prof. Richard Peto

applied to large groups of patients several novel methods of analysis. His chief concern has been that unrealistic hopes of obtaining large treatment effects in clinical trails lead investigators to undervalue and thus to disregard beneficial effects of a more moderate size. By performing large overviews of numerous trials, called "meta-analyses", he has been able to prove that particular therapies are effective or ineffective. In fact, his results have revolutionized the treatment of heart attack, the use of aspirin to prevent strokes, and the use of certain drugs for the treatment of breast cancer.

Finally, Dr. John R. Evans, the Chairman of Allelix Biopharmaceuticals Inc., was awarded The Gairdner Foundation Wightman Award. This particular award



Dr. John R. Evans

was established in 1976 to recognize Canadians who have shown outstanding leadership in medicine and medical science. Dr. Evans proved worthy of such an award by distinguishing himself in five medically-related careers. He has excelled in health research, health professional education, university governance, international health, and the health research industry. He trained at Toronto, Oxford, and Harvard as a clinical investigator of myocardial metabolism. Later, he was the founding Dean of the Faculty of Medicine at McMaster (1965-72), where he led a revolution in problem-based, self-directed medical education which has been replicated at health science centres on every continent. In addition, he was the President of the University of Toronto from 1972 to 1978, where he introduced major changes in university governance and strengthened the Faculty of Medicine. As Founding Director of the Department of Population, Health, and Nutrition of the World Bank (1979-1983), and later as a Trustee of the Rockefeller Foundation, he became a leader in effecting original programmes of health policy, health professional education and health research in developing countries of the world. His fifth and present career is as Chair and CEO of Allelix, a leading biotechnology research and development company.

The Gairdner Foundation, established in 1957 by the late James A. Gairdner, has honoured 225 scientists over the last 35 years. Forty of these have then gone on to receive a Nobel Prize. Mr. Gairdner was born in Toronto in 1893. He served in the Canadian army in World War I but retired from the forces in 1918 as a Major to begin a successful career as a stockbroker and industrialist. At the age of 56, after suffering a severe attack of arthritis, he took up oil painting. In fact, Gairlock Gardens in Oakville, the place where he lived and painted, is now an art gallery and public park.

Throughout his life, James Gairdner showed a practical interest in clinical medicine and medical research. He also believed strongly that the accomplishments of medical scientists should be recognized. Thus, it was with the encouragement of Dr. Wallace Graham, a leading rheumatologist in Toronto, that he established the Gairdner Foundation International Awards. Originally, these awards were confined to the fields of arthritis, rheumatism and heart disease. More recently, the awards have been extended to include other areas of medicine such as immunology and molecular biology. Although Mr. Gairdner died in 1971, outstanding medical scientists continue to be honoured each year in his name.

We are thankful to the Gairdner Foundation for much of the above information.

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Abstract - An abstract of not more than 200 words, typed on a separate page, is required for each article or note. No abbreviations should appear in the abstract. A translation of the abstract into French, if possible, should be included (abstract only).

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The reference list should be placed at the end of the text beginning on a separate sheet and should be listed with the arabic numeral following the order of appearance in the text. Journal article references should correspond to the following style:

You Ch, Lee Ky, Chey Wy, Menguy R. Electogastrographic study: a patient with unexplained nausea, bloating and vomiting. Gastroenterology 1980; 79:311-314.

For further details, see CMAJ 1985;133:443-448.

Tables - Tables should be numbered with arabic numerals, have a brief title, and be referred to in the text. Column headings and descriptive matter in tables should be brief. Vertical rules should not be used.

Illustrations - Each figure should be planned to fit into the area of either one or two columns of text. Photographs and illustrations must be black and white and of good quality. Colour will only be considered if the author, or an outside sponsor, is willing to pay the cost. The figures (including halftones, photographs) are numbered consecutively in arabic numerals, and each one must be referred to in the text as well as being self-explanatory. Only essential labelling should be used with detailed information given in the caption. Letters and numerals should be of such size that the smallest character will not be less than 1 mm high when reduced.

Drug Names -Both nonproprietry (generic) and trade names should be provided for all drugs mentioned.

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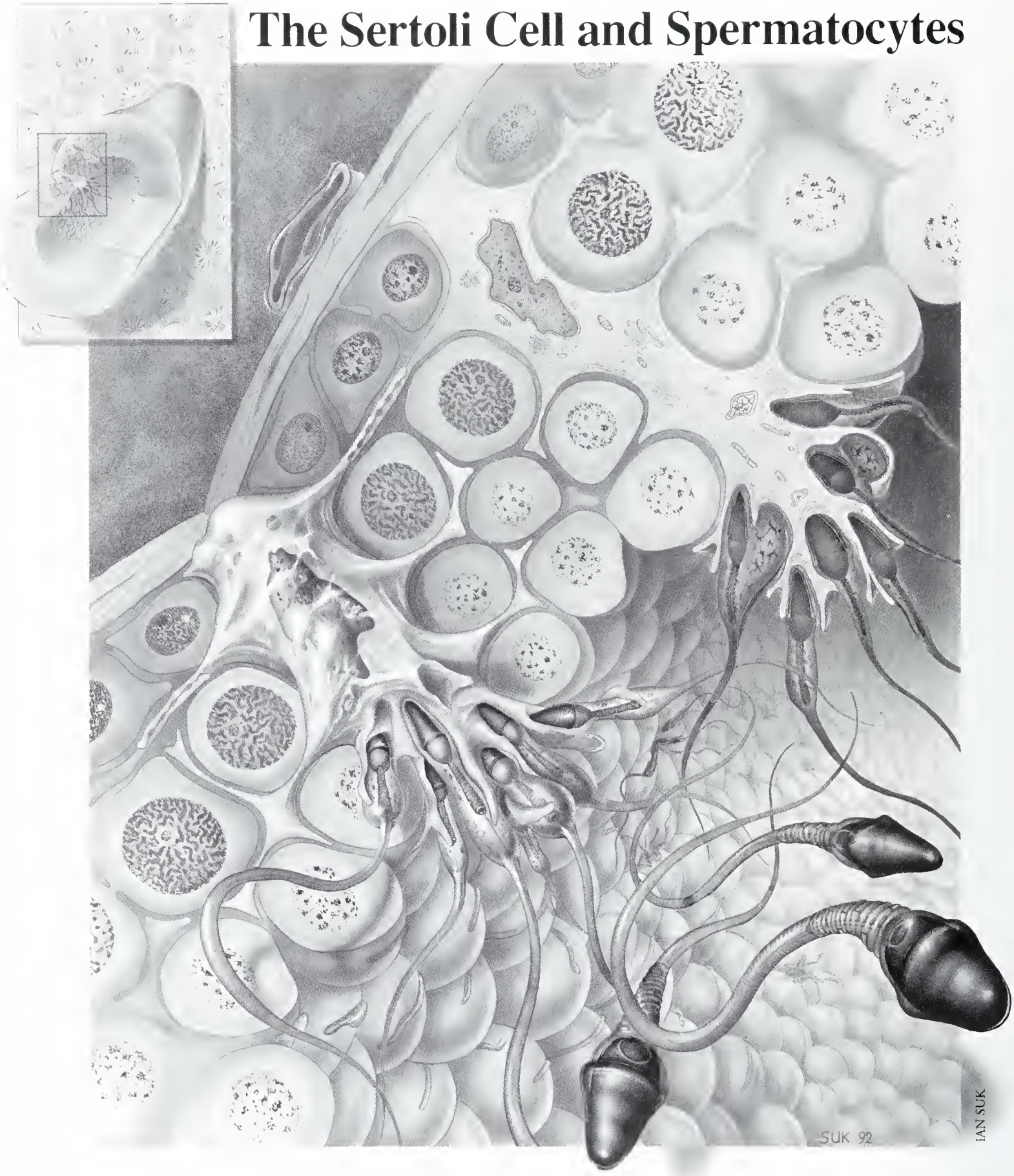
The Sertoli Cell and Spermatocytes



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